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Proposed Re-evaluation Decision PRVD2015-05

d-Phenothrin

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Overview

What Is the Proposed Re-evaluation Decision?

After a re-evaluation of the insecticide d-phenothrin, Health Canada's Pest Management Regulatory Agency (PMRA), under the authority of the *Pest Control Products Act* and Regulations, is proposing continued registration for the use and sale of d-phenothrin products in Canada.

An evaluation of available scientific information found that products containing d-phenothrin do not present unacceptable risks to human health or the environment when used according to the proposed label directions. As a condition of the continued registration of d-phenothrin, new risk-reduction measures are proposed for the end-use products registered in Canada. No additional data are being requested at this time.

This proposal affects all end-use products containing d-phenothrin registered in Canada. Once the final re-evaluation decision is made, registrants will be instructed on how to address any new requirements.

This Proposed Re-evaluation Decision is a consultation document[1] that summarizes the science evaluation for d-phenothrin and presents the reasons for the proposed re-evaluation decision. It also proposes new risk-reduction measures to further protect the environment.

This consultation document is presented in two parts. The Overview describes the regulatory process and key points of the evaluation, while the Science Evaluation provides detailed technical information on the assessment of d-phenothrin.

The PMRA will accept written comments on this proposal up to 45 days from the date of publication of this document. Please forward all comments to Publications (please see contact information indicated on the cover page of this document).

What Does Health Canada Consider When Making a Re-evaluation Decision?

The PMRA pesticide re-evaluation program considers potential risks, as well as value, of pesticide products to ensure they meet modern standards established to protect human health and the environment. Regulatory Directive DIR2012-02, *Re-evaluation Program Cyclical Re-evaluation*, presents the details of the current re-evaluation approach.

For more details on the information presented in this overview, please refer to the Science Evaluation section of this consultation document.

[1] "Consultation statement" as required by subsection 28(2) of the *Pest Control Products Act*.

What Is d-Phenothrin?

The insecticide d-phenothrin is a synthetic pyrethroid insecticide used to control a broad range of arthropod pests on a wide variety of sites, including in and around structures, indoor and outdoor ornamental plants and mattresses. This insecticide is also used as flea and tick control products. It works by contact and stomach action, and is fast acting. It is applied by members of the general public and professional applicators using an applicator tube, pressurized spray can or shaker can.

Health Considerations

Can Approved Uses of d-Phenothrin Affect Human Health?

The insecticide d-phenothrin is unlikely to affect your health when used according to label directions.

Potential exposure to d-phenothrin (sumithrin technical grade) may occur through the diet (food and water), when handling and applying the product, or when entering treated sites. When assessing health risks, two key factors are considered: the levels at which no health effects occur and the levels to which people may be exposed. The dose levels used to assess risks are established to protect the most sensitive human population (for example, children and nursing mothers). Only uses for which the exposure is well below levels that cause no effects in animal testing are considered acceptable for registration.

Toxicology studies in laboratory animals describe potential health effects from varying levels of exposure to a chemical and identify the dose at which no effects are observed. The health effects noted in animals occur at doses more than 100 times higher (and often much higher) than levels to which humans are normally exposed when pesticide-containing products are used according to label directions.

In laboratory animals, the technical grade active ingredient d-phenothrin was slightly acutely toxic by the inhalation route of exposure. It was of low acute toxicity by the oral and dermal routes, minimally irritating to the eyes, not irritating to the skin, and did not cause an allergic skin reaction.

There was no evidence to suggest that d-phenothrin damaged genetic material and it is not considered to be a potential human carcinogen. Although d-phenothrin exerts its action on the nervous system, there was little evidence of neurotoxicity. There was no indication that d-phenothrin caused damage to the immune system or affected the ability to reproduce. Health effects in animals given repeated doses of d-phenothrin included effects on the liver, adrenals and kidneys.

When given to pregnant or nursing rats, d-phenothrin caused slight, transient decreases in body weight of the young animal at doses that were not toxic to the mother, suggesting that the young were slightly more sensitive to d-phenothrin than the adult animal. Effects on the developing fetus (malformations) were noted following administration of d-phenothrin to pregnant rabbits. These effects occurred at doses that were also toxic to the mother.

The risk assessment protects against the effects of d-phenothrin by ensuring that the level of human exposure is well below the lowest dose at which these effects occurred in animal tests.

Residues in Food and Drinking Water

Dietary risks from food and water are not of concern.

Reference doses define levels to which an individual can be exposed over a single day (acute) or lifetime (chronic) and expect no adverse health effects. Generally, dietary exposure from food and water is acceptable if it is less than 100% of the acute reference dose or chronic reference dose (acceptable daily intake). An acceptable daily intake is an estimate of the level of daily exposure to a pesticide residue which, over a lifetime, is believed to have no significant harmful effects.

There are no agricultural uses of d-phenothrin in Canada. The only possible source of residue in the Canadian diet would be from imported food commodities from indirect applications to food commodities.

Acute and chronic dietary exposures to d-phenothrin were estimated from potential residues of d-phenothrin from imported commodities. Exposures were assessed for different subpopulations including children and women of reproductive age.

The acute dietary exposure estimate (from food and drinking water) at the 99.9th percentile was less than 1% of the acute reference dose for the general population and for all population subgroups. The chronic dietary exposure estimate for the general population and for all population subgroups was less than 1% of the acceptable daily intake. Thus, acute and chronic dietary risks are not of concern.

The *Food and Drugs Act* prohibits the sale of adulterated food, that is, food containing a pesticide residue that exceeds the established maximum residue limit. Pesticide maximum residue limits are established through the evaluation of scientific data under the *Pest Control Products Act*. Each maximum residue limit value defines the maximum concentration in parts per million (ppm) of a pesticide allowed in or on certain foods. Food containing a pesticide residue that is at or below the established maximum residue limit does not pose a health risk concern.

Since there are no Canadian or international food uses of d-phenothrin, maximum residue limits have not been specified. Where no specific maximum residue limit has been established, subsection B.15.002(1) of the Food and Drug Regulations applies, which requires that residues not exceed 0.1 ppm. Additional details regarding maximum residue limits can be found in the Science Evaluation section of this consultation document.

Risks in Residential and Other Non-Occupational Environments from d-Phenothrin

Non-occupational risks are not of concern when d-phenothrin is used according to label directions.

Residential exposure may occur from the application of products containing d-phenothrin to residential gardens and trees, indoor environments, and pets. Homeowner exposure would occur from applying domestic-class d-phenothrin products. These products can be applied by aerosol can, shaker can and spot-on treatments (pets).

Residential postapplication exposure may occur while performing activities on treated areas. Treated areas include areas treated by residential handlers as well as residential areas treated by commercial applicators. Exposure would be predominantly dermal and by inhalation. Incidental oral exposure may also occur for children (1 to < 2 years old) playing in treated areas or in contact with treated dogs.

For all domestic-class products, the target dermal and inhalation margins of exposure (MOEs) were met for adults applying d-phenothrin and are not of concern. Residential postapplication activities also met the target dermal and inhalation MOEs for all populations and are not of concern. For incidental oral exposure, the target oral MOE was met for children (1 to < 2 years old) and are not of concern.

Incidental oral scenarios were aggregated with background (chronic) dietary exposure (food and drinking water). The resulting aggregate risk estimates reach the target MOE for all uses and are not of concern.

Occupational Risks from d-Phenothrin

Occupational risks are not of concern when d-phenothrin is used according to label directions.

The calculated dermal and inhalation MOEs are greater than the target MOE for all of the commercial applicator scenarios using baseline personal protective equipment. As such, no additional mitigation measures are required for these scenarios. The MOEs were calculated using the highest application rate of all of the commercial products.

It was assumed that risks to postapplication workers would be similar to or less than residential postapplication risks. As no risks of concern were identified for residential postapplication scenarios, a specific assessment for postapplication workers was not required.

Environmental Considerations

What Happens When d-Phenothrin Is Introduced Into the Environment?

The insecticide d-phenothrin is used primarily in and around homes as a domestic insecticide. Based on this use pattern, environmental exposure is expected to be minimal. It can enter soil and surface water if released into the environment. This insecticide is non-persistent in soil, breaking down in the presence of microbes. In water, d-phenothrin is broken down rapidly by sunlight and microbes and, consequently, is not expected to persist in aquatic environments. The insecticide d-phenothrin is not expected to enter the atmosphere and be subject to long-range transport. Laboratory studies indicate that d-phenothrin is not likely to move downward through the soil, indicating that it has a low potential to leach to ground water.

The insecticide d-phenothrin poses negligible risk to terrestrial birds and mammals. At high enough doses, it can be toxic to terrestrial and aquatic invertebrates and fish. However, due to its use pattern, the potential exposure of terrestrial and aquatic non-target organisms is expected to be minimal; consequently, the risk to these organisms is not of concern.

Proposed Measures to Minimize Risk

Labels of registered pesticide products include specific instructions for use. Directions include risk-reduction measures to protect human health and the environment. These directions must be followed by law. As a result of the re-evaluation of d-phenothrin, the PMRA is proposing further risk-reduction measures for product labels.

Human Health

As no risks of concern were identified for d-phenothrin, no additional mitigation measures are required. However, revisions to the labels are proposed to provide consistency across co,mmon products, update label statements and minimize unnecessary exposure. The proposed label amendments are listed in Appendix VIII.

Environment

Due to the limited outdoor use of d-phenothrin, the risk to terrestrial and aquatic organisms is expected to be minimal. However, precautionary statements are being proposed to further protect the environment. The proposed label amendments are listed in Appendix VIII.

Next Steps

Before making a final re-evaluation decision on d-phenothrin, the PMRA will consider all comments received from the public in response to this consultation document. A science-based approach will be applied in making a final decision on d-phenothrin. The PMRA will then publish a Re-evaluation Decision[2] that will include the decision, the reasons for it, a summary of comments received on the proposed decision and the PMRA's response to these comments.

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2 "Decision statement" as required by subsection 28(5) of the *Pest Control Products Act*.

Science Evaluation

1.0 Introduction

The insecticide *d*-phenothrin is under re-evaluation in Canada as described by the Pest Management Regulatory Agency (PMRA) in the December 20, 2011 Re-evaluation Note REV2011-05, *Re-evaluation of Pyrethroids, Pyrethrins and Related Active Ingredients*. It is a broad spectrum contact synthetic pyrethroid belonging to the Insecticide Resistance Management Mode of Action (MoA) group 3A.

Following the re-evaluation announcement for *d*-phenothrin, the registrant of the technical grade active ingredient, and primary data provider in Canada indicated continued support for all registered label uses.

Currently registered products containing *d*-phenothrin are listed in Appendix I. All current uses are being supported by the registrant and were, therefore, considered in the re-evaluation of *d*-phenothrin.

The purpose of this re-evaluation is to review existing information on the active ingredient, *d*-phenothrin, and the currently registered *d*-phenothrin technical, commercial-class and domestic-class end-use products, to ensure that risk assessments meet current standards.

2.0 The Technical Grade Active Ingredient, Its Properties and Uses

2.1 Identity of the Technical Grade Active Ingredient

Common Name	There is no ISO common name for *d*-phenothrin (4:1 mixture of the [1R,*trans*] and [1R,*cis*] isomers). Phenothrin is the ISO common name for the racemic mixture of 4 stereoisomers.
Function	Insecticide
Chemical Family	Pyrethroid

Chemical Name

1	**International Union of Pure and Applied Chemistry**	(RS) (3-phenoxyphenyl)methyl (1*E*,3*E*)-2,2-dimethyl-3-(2-methylprop-1-en-1-yl)cyclopropane-1-carboxylate OR 3-phenoxybenzyl (1*RS*,3*RS*;1*RS*,3*SR*)-2,2-dimethyl-3-(2-methylprop-1-enyl)cyclopropanecarboxylate OR 3-phenoxybenzyl (1*RS*)-*cis*-*trans*-2,2-dimethyl-3-(2-methylprop-1-enyl)cyclopropanecarboxylate OR 3-phenoxybenzyl (±)-*cis*-*trans*-chrysanthemate
2	**Chemical Abstracts Service (CAS)**	(3-Phenoxyphenyl)methyl 2,2-dimethyl-3-(2-methyl-1-propenyl)-cyclopropanecarboxylate

CAS Registry Number 26002-80-2 for the racemic mixture

Molecular Formula

Structural Formula $C_{19}H_{20}O_3$

Molecular Weight 350.46

Purity of the Technical Grade Active Ingredient 95.6%

2.2 Physical and Chemical Properties of the Technical Grade Active Ingredient

Property	Result
Vapour pressure at 21.4°C	1.9×10^{-7} mPa
Ultraviolet/visible spectrum	solvent: lumax (nm) ethanol: 270, 279
Solubility in water at 25°C	< 0.7 µg/L
n-Octanol/water partition coefficient at 25°C	log K_{ow} = 6.01
Dissociation constant	Product does not contain any dissociable moiety.

2.2 Description of Registered d-Phenothrin Uses

Appendix IIA lists all commercial class uses for which d-phenothrin is currently registered, while Appendix IIB lists all domestic class uses for which d-phenothrin is currently registered.

Uses of d-phenothrin belong to the following use-site categories: structures, companion animals, human skin, clothing and personal use, outdoor ornamentals, indoor plants and plantscapes, and residential outdoors.

3.0 Impact on Human and Animal Health

3.1 Toxicological Summary

A detailed review of the toxicological database for the technical grade active ingredient d-phenothrin was conducted. The database was considered complete and contained all the full array of toxicity studies currently required for hazard assessment purposes. The toxicology database supporting d-phenothrin was primarily based on studies available from the registrant and it was considered adequate to define the majority of the toxic effects that may result from exposure.

The insecticide d-phenothrin is a type I synthetic pyrethroid insecticide. Pyrethroids delay the closing of neuronal voltage-dependent sodium channels, causing the depolarization of the neuron. This interferes with the ability of the nervous system to relay nerve transmissions and may result in downstream clinical effects. Technical d-phenothrin is predominantly a mixture of 1R, *cis*- and 1R, *trans*-isomers with a typical *cis*-to-*trans* ratio of 25:75.

Radiolabelled d-phenothrin was rapidly absorbed and distributed following either single or repeated oral exposure in the rat. Tissue levels of radioactivity were low under all of the dosing regimens. Peak tissue levels were noted three hours post-dosing, with most of the radioactivity disappearing within 24 hours following dosing. Residues of d-phenothrin were primarily located in the fat, with levels of radioactivity in fat associated with the *trans*-isomer being lower than those identified with the *cis*-isomer. For both isomers, levels of radioactivity were higher following repeated oral doses than following a single dose. There was little evidence of bioaccumulation potential with either of the dosing regimens.

With both isomers of d-phenothrin, major metabolites identified in both sexes of rat were 4'-hydroxyl-phenoxybenzoic acid (4HBA) and 3-phenoxybenzoic acid. Urinary metabolites were derived following ester cleavage of the parent compound, whereas fecal metabolites of the *cis*-isomer retained the ester linkage and were derived from oxidation. The metabolite pattern appeared to be the same regardless of the dose regimen and no sex-related differences were apparent.

After administration of single doses of either isomer in rats, the primary route of excretion was the feces, with higher fecal excretion noted for the *cis*-isomer. After repeated dosing, the fecal route was still the predominant route for the *cis*-isomer whereas urinary excretion was the predominant route of elimination for the *trans*-isomer. Following either single or repeated dosing of d-phenothrin in rats, excretion was virtually complete within seven days of dosing. There was no detectable radioactivity in the expired air of rats treated with single or repeated oral doses of the *cis*- or *trans*-isomer of d-phenothrin.

In rats, technical d-phenothrin was of low acute toxicity by the oral and dermal routes and slightly toxic by the inhalation route of exposure. The insecticide d-phenothrin was minimally irritating to the eyes of rabbits but was not a skin irritant in rabbits or a potential skin sensitizer in the maximization test when tested with guinea pigs. Clinical signs of toxicity were noted only in the acute inhalation toxicity studies and included slight excitation, laboured breathing, rales, nasal discharge, dried red material on the facial area, chromodacryorrhea and urinary incontinence.

Repeated oral and inhalation administration of d-phenothrin to mice, rats and dogs revealed that the target organ was the liver, with increased organ weights noted at lower dosage levels followed by increased enzyme levels and histopathology (hepatocellular hypertrophy, coagulative liver necrosis and bile duct proliferation) at higher dosage levels. Toxicologically significant effects on the adrenal gland (organ weight increases and histopathological alterations) and the kidney (increased organ weights and alterations in clinical chemistry) were also noted at higher dosage levels. Additional effects seen in the 90-day inhalation study in rats included eosinophilic inclusions in the olfactory epithelial cells of the nasal turbinates and clinical signs of toxicity (poorly groomed fur, dirty tails and a reduced response to a knock on the chamber door). In a 21-day dermal toxicity study, no signs of systemic toxicity were noted in rats receiving the limit dose of 1000 mg/kg bw/day. Based on the results of these repeat-dose toxicity studies, the dog appeared to be the most sensitive species. As well, it was noted that prolonging the duration of exposure (from intermediate to chronic durations) to d-phenothrin by the oral route led to increased toxicity in rats and dogs.

In a two-year dietary chronic toxicity/carcinogenicity study in mice, exposure to d-phenothrin did not result in any overt signs of toxicity or effects on survival. An increased incidence of hepatocellular adenomas was noted in both sexes while an increased incidence of hepatocellular carcinomas was noted only in females exposed to d-phenothrin. The increased incidence of hepatocellular adenomas noted in males was slightly above the historical control range provided by the registrant but did not demonstrate any progression to hepatocellular carcinomas. In females, the incidence of hepatocellular adenomas was within the historical control range; however, the incidence of hepatocellular carcinomas was only slightly above the historical control range at the high-dose level. Given that the increase in the combined incidence of adenomas and carcinomas in female mice was not statistically significant and only marginally exceeded the historical control range, the level of concern was considered low for these tumours.

In rats, a two-year dietary chronic toxicity/carcinogenicity study demonstrated a marginally increased incidence of adenomas and carcinomas of the preputial gland at a dosage level of 141 mg/kg bw/day. In a second rat carcinogenicity study, there was no evidence of preputial

tumours, even at significantly greater dosage levels. In view of these findings, the overall weight of evidence suggested a low level of concern for preputial gland tumours in rats. In the second two-year study, an increased incidence of hepatocellular adenomas and carcinomas was noted in high-dose males and females along with an increased incidence of uterine adenomas and adenocarcinomas in high-dose females. The increased incidence of tumours at the high-dose level was of limited concern given the fact that this dosage level resulted in severe liver toxicity and clearly exceeded the maximum tolerated dose.

The insecticide d-phenothrin showed no evidence of mutagenicity, with or without metabolic activation, in the in vitro bacterial/microsomal reverse mutation studies with *Salmonella typhimurium* or *Escherichia coli*. Negative results were also noted in an in vitro unscheduled DNA synthesis assay in HeLa S3 human cells. No evidence of clastogenic potential was noted in an in vitro chromosomal aberration assay conducted with Chinese hamster ovary cells, or in an in vivo assay conducted with the bone marrow cells of importing control region mice (ICR mice).

Two multi-generation dietary reproduction toxicity studies were conducted with rats. In the first of these studies, effects on the development of the offspring were noted only at dosage levels resulting in maternal toxicity. Therefore, sensitivity of the young was not demonstrated in this study. In the more recent two-generation rat reproductive toxicity study, decreased pup weight was noted in the second generation offspring from postnatal days 1 to 14, in the absence of maternal toxicity. At higher dosage levels, more significant effects in the pups (such as effects on survival) were noted in the presence of severe maternal toxicity. Results of this study suggested sensitivity of the young; however, given that the effect on pup body weight occurred only in the second generation, was transient and of a small magnitude, the concern for sensitivity was low.

In a developmental toxicity study in rats, oral gavage administration of d-phenothrin resulted in reduced maternal and fetal body weights, delayed ossification and fetal immaturity as demonstrated by dilation of the brain ventricles and space between the body wall and organs at the same dose levels. Since developmental effects were observed only at maternally toxic dose levels, it was concluded that increased susceptibility of the young was not demonstrated through in utero exposure.

In an oral gavage rabbit developmental toxicity study, maternal toxicity in the form of weight loss, decreased body weight gain and food consumption was noted starting at 300 mg/kg bw/day. An increased number of abortions were observed at the highest dose tested of 500 mg/kg bw/day. At 500 mg/kg bw/day, four fetuses from three separate litters displayed hydrocephaly; the incidence exceeded historical control data and was considered treatment-related. Sensitivity of the young was not demonstrated in rabbits as the effects in the developing fetus were observed only in the presence of maternal toxicity.

In an acute neurotoxicity study conducted via the oral route with rats, no clinical signs of toxicity, effects on motor activity, or adverse histopathology were noted. Similarly, in a 13-week oral neurotoxicity study, no evidence of neurotoxicity was noted in rats. The effects that were noted in this repeat-dose study were limited to decreased body weight, body weight gain and food consumption; however, it should be noted that only a limited number of parameters are examined in studies of this type. The d-phenothrin toxicity database as a whole showed little to no evidence of neurological signs typically associated with pyrethroids.

Uterotrophic and Hershberger assays with d-phenothrin were reported in the published literature. Based on the results of these two in vivo assays, it was concluded that d-phenothrin did not exhibit any potential to cause adverse estrogenic or (anti-) androgenic effects at dosage levels up to and including 1000 mg/kg bw/day.

Despite a lack of strong evidence of increased sensitivity of the offspring in any of the submitted studies, residual uncertainty remains regarding susceptibility of the young. Literature studies indicate that pharmacodynamic and pharmacokinetic factors, notably age-dependent maturation of key metabolic processes, may lead to increased susceptibility of the young to pyrethroid toxicity. Young animals have incomplete maturation of the enzyme systems that detoxify pyrethroids, particularly the carboxylesterases and cytochrome P450s. Consequently, pyrethroid concentrations in target tissues (such as the brain) may be higher in young animals than in adults given the same dose. In general, pyrethroid neurotoxicity is correlated to peak concentrations of the compound, with gavage-dosing patterns resulting in greater internal doses compared to dietary administration. The pyrethroids are regarded as having a narrow window of time-to-peak effect. The design of a developmental neurotoxicity study does not consider time-to-peak effect and may miss the window of peak toxicity for the pyrethroids (US EPA, 2010). Accordingly, a developmental neurotoxicity study is not required for d-phenothrin.

Behavioural assessments were conducted at the time-to-peak effect in adults in an acute neurotoxicity study with d-phenothrin; however, behavioural assessments were not conducted in offspring. In the recent evaluation of other pyrethroids, a similar situation has resulted in the application of a database uncertainty factor for the lack of a comparative oral gavage neurotoxicity study considering time-to-peak effect in pups, weanlings and adult animals. This factor has not been applied in the case of d-phenothrin in view of the lack of neurotoxicity noted in the animal toxicity data. Endpoints selected for risk assessments were well below the dose levels employed and without effect in adult animals in the acute neurotoxicity study, thus affording intrinsic protection to the young for potential neurotoxicity.

Results of the toxicology studies conducted on laboratory animals with d-phenothrin, along with the toxicology endpoints for use in the human health risk assessment, are summarized in Tables 1 and 2 of Appendix III.

3.1.1 *Pest Control Products Act* Hazard Characterization

For assessing risks from potential residues in food or from products used in or around homes or schools, the *Pest Control Products Act* requires the application of an additional 10-fold factor to threshold effects to take into account completeness of the data with respect to the exposure of and toxicity to infants and children, and potential prenatal and postnatal toxicity. A different factor may be determined to be appropriate on the basis of reliable scientific data.

With respect to the completeness of the toxicity database as it pertains to the exposure of and toxicity to infants and children, extensive data were available for d-phenothrin. The database contains the full complement of required studies including developmental toxicity studies in rats and rabbits, and two multi-generation reproductive toxicity studies in rats. A comparative neurotoxicity study was not available. But given the lack of neurotoxic findings throughout the d-phenothrin database, the results of such a study in this case would not be expected to significantly affect the risk assessment.

With respect to potential prenatal and postnatal toxicity, decreased pup weight was noted in the second generation of the two-generation rat reproduction toxicity study in the absence of maternal toxicity. At higher dosage levels, more significant effects in the pups (such as effects on survival) were noted in the presence of severe maternal toxicity. Results of this study demonstrated sensitivity of the young; however, there was a low level of concern given the fact that the magnitude of the effect on the body weight of the pups was slight and recovery was noted prior to weaning of the pups.

In a developmental toxicity study in rats, there was an increased incidence of delayed ossification and fetal immaturity as demonstrated by dilation of the brain ventricles and space between the body wall and organs, in the presence of maternal toxicity. In a rabbit developmental study, an increased incidence of abortions and malformations (hydrocephaly) was noted at a maternally toxic dosage level.

Overall, the database is adequate for determining the sensitivity of the young, and effects on the young are well characterized. Although the fetal effects in the rabbit developmental toxicity study were considered serious endpoints, the concern was tempered by the presence of maternal toxicity suggesting that a threefold *Pest Control Products Act* factor would be required. Since the selected endpoints for risk assessment provide an intrinsic margin to the malformations, the *Pest Control Products Act* factor has been reduced to onefold.

3.1.2 Cancer Assessment

Since there was no clear evidence of carcinogenicity at doses below the maximum tolerated dose, a cancer risk assessment was not conducted.

3.2 Dietary Exposure and Risk Assessment

In a dietary exposure assessment, the PMRA determines how much of a pesticide residue, including residues in milk and meat, may be ingested with the daily diet. Exposure from potential residues in imported foods is also included in the assessment. These dietary assessments are age specific and incorporate the different eating habits of the population at various stages of life (infants, children, adolescents, adults and seniors). For example, the assessments take into account differences in children's eating patterns, such as food preferences and the greater consumption of food relative to their body weight when compared to adults. Dietary risk is then determined by the combination of the exposure and the toxicity assessments. High toxicity may not indicate high risk if the exposure is low. Similarly, there may be risk from a pesticide with low toxicity if the exposure is high.

The PMRA considers limiting use of a pesticide when risk exceeds 100% of the reference dose. The PMRA Science Policy Note SPN2003-03, *Assessing Exposure from Pesticides, A User's Guide*, presents detailed acute, chronic and cancer risk assessment procedures.

Residue estimates used in the dietary risk assessment may be based conservatively (upperbound estimates) using MRLs, or the field trial data representing the residues that may remain on food after treatment at the maximum label rate. Surveillance data representative of the national food supply may also be used to derive a more accurate estimate of residues that may remain on food when it is purchased. These include the Canadian Food Inspection Agency's National Chemical Residue Monitoring Program and the United States Department of Agriculture's Pesticide Data Program (USDA-PDP). Specific and empirical processing factors, as well as specific information regarding percent of crops treated, may also be incorporated to the greatest extent possible.

In situations where the need to mitigate dietary exposure has been identified, the following options are considered. Dietary exposure from Canadian agricultural uses can be mitigated through changes in the use pattern. Revisions of the use pattern may include such actions as reducing the application rate or the number of seasonal applications, establishing longer pre-harvest intervals, and/or removing uses from the label. In order to quantify the impact of such measures, new residue chemistry studies that reflect the revised use pattern would be required. These data would also be required in order to amend MRLs to the appropriate level. Imported commodities that have been treated also contribute to the dietary exposure and are routinely considered in the risk assessment. The mitigation of dietary exposure that may arise from treated imports is generally achieved through the amendment or specification of MRLs.

Acute and chronic exposure and risk assessments for d-phenothrin were conducted using the Dietary Exposure Evaluation Model – Food Commodity Intake Database™ (DEEM-FCID™, Version 2.14), which incorporates consumption data from the United States Department of Agriculture's Continuing Surveys of Food Intakes by Individuals from 1994 to 1996 and 1998. Acute and chronic exposure and risk estimates for d-phenothrin appear in Tables 1 and 2, Appendix V.

There are no agricultural uses of d-phenothrin in Canada. The only possible source of residue in the Canadian diet would be from imported food commodities from indirect applications to foods. The residue chemistry of d-phenothrin is summarized in Appendix VI.

3.2.1 Acute Reference Dose

To estimate acute dietary risk (1 day) for the general population, an oral developmental toxicity study in the rabbit was selected for risk assessment. A no observed adverse effect level (NOAEL) of 100 mg/kg bw/day was selected based on weight loss, decreased body weight gain and food consumption at the next dosage level, starting from as early as gestation day 7. Given that these alterations were noted after a single exposure of d-phenothrin, this study was considered relevant in the establishment of an acute reference dose. Standard uncertainty factors of 10-fold for interspecies extrapolation and 10-fold for intraspecies variability were applied. The *Pest Control Products Act* factor was reduced to onefold as discussed in the *Pest Control Products Act* Hazard Characterization section resulting in a composite assessment factor (CAF) of 100.

$$\textbf{ARfD} = \frac{\textbf{NOAEL}}{\textbf{CAF}} = \frac{\textbf{100 mg/kg bw/day}}{\textbf{100}} = \textbf{1.0 mg/kg bw/day}$$

The acute reference dose (ARfD) provides a margin of 300 to the NOAEL for developmental toxicity in the rabbit and is thus considered protective of all populations including pregnant women and their fetuses, infants and children.

3.2.2 Acute Dietary Exposure and Risk Assessment

The acute dietary risk was calculated considering the highest ingestion of d-phenothrin that would be likely on any one day, and using food consumption and food residue values. The expected intake of residues is compared to the ARfD, which is the dose at which an individual could be exposed on any given day and expect no adverse health effects. When the expected intake of residues is less than the ARfD, then acute dietary exposure is not of concern.

Acute exposure estimates were determined for the general population and other population subgroups. This was achieved by assuming zero residues in Canadian food commodities, and using USDA-PDP monitoring data for foods imported into Canada from the United States, where indirect application to foods may occur during mosquito control abatement programs. The United States tolerance of 0.01 ppm was assumed for all other crops from the United States, which were not surveyed in the USDA-PDP. Default processing factors, the assumption that 100% of imported crops are treated, and Canadian import and production statistics were also used in the assessment. Drinking water contribution from Canadian uses was assumed to be zero as there are no agricultural uses for d-phenothrin (see Section 3.3).

The acute dietary exposure estimate (from food and drinking water) at the 99.9th percentile was less than 1% of the ARfD for the general population and for all population subgroups. Thus, acute dietary risks are not of concern.

3.2.3 Acceptable Daily Intake

To estimate dietary risk from repeated exposure for the general population, the 12-month dog dietary study with a NOAEL of 7.1 mg/kg bw/day was selected for risk assessment purposes. At the lowest observed adverse effect level (LOAEL) of 26.8 mg/kg bw/day, pituitary microcysts were noted in both males and females. Focal degeneration of the adrenal cortex with mononuclear cell infiltration of the adrenal glands, diffuse hepatocellular enlargement and focal mononuclear infiltration of the epididymides were noted in males. This study provides the lowest NOAEL in the database, uses the most sensitive species and since the available toxicology database suggests that increased duration of oral exposure (intermediate to chronic) increases the toxicity of d-phenothrin, this study is also of appropriate duration for setting this chronic reference dose. Standard uncertainty factors of 10-fold for interspecies extrapolation and 10-fold for intraspecies variability have been applied. As discussed in the *Pest Control Products Act* Hazard Characterization section, the *Pest Control Products Act* factor was reduced to onefold, resulting in a CAF of 100.

$$\textbf{ADI} = \frac{\textbf{NOAEL}}{\textbf{CAF}} = \frac{\textbf{7.1 mg/kg bw/day}}{\textbf{100}} = \textbf{0.07 mg/kg bw/day}$$

The acceptable daily intake (ADI) provides a margin of > 4200 to the NOAEL for developmental toxicity in the rabbit and is thus considered protective of all populations including pregnant women and their fetuses, infants and children.

3.2.4 Chronic Dietary Exposure and Risk Assessment

The chronic dietary risk was calculated by using the average consumption of different foods and the average residue values on those foods. This expected intake of residues was then compared to the ADI. When the expected intake of residues is less than the ADI, then chronic dietary exposure is not of concern.

Similar to the acute assessment, chronic exposure estimates were determined for the general population and other population subgroups by assuming zero residues in Canadian food commodities, and using USDA-PDP monitoring data for foods imported into Canada from the United States, where indirect application to foods may occur during mosquito control abatement programs. The United States tolerance of 0.01 ppm was assumed for all other crops from the United States, which were not surveyed in the USDA-PDP. Default processing factors, the assumption that 100% of imported crops are treated, and Canadian import and production statistics were also used in the assessment. Drinking water contribution from Canadian uses was assumed to be zero as there are no agricultural uses for d-phenothrin (see Section 3.3).

The chronic dietary exposure from food and drinking water for the general population and for all population subgroups was less than 1% of the ADI. Thus, chronic dietary risks are not of concern.

3.3 Exposure from Drinking Water

3.3.1 Concentrations in Drinking Water

As there are no agricultural uses for d-phenothrin, the agricultural scenarios used in the models for drinking water would not be applicable to the use pattern of the chemical. Drinking water modelling was not conducted.

The PMRA assumed zero residues in drinking water.

3.3.2 Drinking Water Exposure and Risk Assessment

Since exposure from drinking water was assumed to be zero, the exposure from food is considered to be the only pathway of dietary exposure. Please refer to Sections 3.2.2 and 3.2.4 for details.

3.4 Occupational and Residential Risk Assessment

Occupational and residential risk is estimated by comparing potential exposures with the most relevant endpoint from toxicology studies to calculate a MOE. This is compared to a target MOE incorporating uncertainty factors protective of the most sensitive subpopulation. If the calculated MOE is less than the target MOE, it does not necessarily mean that exposure will result in adverse effects, but mitigation measures to reduce risk would be required.

3.4.1 Toxicology Endpoint Selection

3.4.1.1 Short- and Intermediate-term Dermal

For occupational and residential short- and intermediate-term dermal risk assessment, the 21-day dermal toxicity study conducted with rats was selected. In this study, there were no treatment-related effects noted at the NOAEL and highest dose tested of 1000 mg/kg bw/day. The target MOE for this scenario was 100, which accounted for a 10-fold uncertainty factor for interspecies extrapolation and a 10-fold uncertainty factor for intraspecies variability. When converted into an oral equivalent dose using a dermal absorption value of 17%, the dermal dose of 1000 mg/kg bw/day provides a margin that is less than the desired 300 to the developmental toxicity endpoints in rabbits. However, since this dermal absorption value is considered to be an over-estimate of absorption, no additional uncertainty factors were deemed necessary. For residential exposures, the *Pest Control Products Act* factor was reduced to onefold. This MOE was considered to be protective of all populations including pregnant women and their fetuses, infants and children.

3.4.1.2 Short- and Intermediate-term Inhalation

For short- and intermediate-term inhalation risk assessment, the NOAEL of 0.104 mg/L (= 26.6 mg/kg bw/day) from the 90-day inhalation toxicity study in rats was selected. This NOAEL was based on eosinophilic inclusions in the olfactory epithelial cells of the nasal

turbinates in males and females at the next highest concentration. A target MOE of 100 was selected for this scenario, which included an uncertainty factor of 10-fold for interspecies extrapolation and a 10-fold factor for intraspecies variability. For residential exposures, the *Pest Control Products Act* factor was reduced to onefold. These values were considered to be protective of all populations including pregnant women and their fetuses, infants and children.

3.4.1.3 Non-dietary Oral Ingestion (Children, Short-term)

For non-dietary oral ingestion risk assessment, the developmental toxicity study in the rabbit was selected. In this study, a NOAEL of 100 mg/kg bw/day was selected based on weight loss, decreased body weight gain and food consumption that occurred at the next dosage level. These effects are endpoints that could result from a short-term exposure and therefore are considered relevant for this scenario. Standard uncertainty factors of 10-fold for interspecies extrapolation and 10-fold for intraspecies variability were applied. As discussed in the *Pest Control Products Act* Hazard Characterization section, the *Pest Control Products Act* factor was reduced to onefold. The target MOE for this scenario was 100.

3.4.1.4 Long-term Dermal, Inhalation and Non-dietary Oral Ingestion

For occupational and residential long-term dermal risk assessment, a NOAEL of 7.1 mg/kg bw/day from the 12-month dog dietary study was selected. At the LOAEL of 26.8 mg/kg bw/day, pituitary microcysts were noted in both males and females, while focal degeneration of the adrenal cortex with mononuclear cell infiltration of the adrenal glands, diffuse hepatocellular enlargement and focal mononuclear infiltration of the epididymides were noted in males. The target MOE for this scenario was 100, which accounted for uncertainty factors of 10-fold for interspecies extrapolation and 10-fold for intraspecies variability. For residential exposures, the *Pest Control Products Act* factor was reduced to onefold. These values were considered to be protective of all populations including pregnant women and their fetuses, infants and children.

3.4.1.5 Dermal Absorption

Dermal absorption is not required for short- to intermediate-term exposure risk assessments as the toxicological endpoint selected is based on a dermal toxicity study. However, a dermal absorption value is required for the long-term exposure risk assessment as the long-term endpoints are based on an oral study. One study was available for the evaluation of dermal absorption. This study demonstrated differential dermal absorption for the *cis*- and *trans*-isomers of 17% and 11% respectively. As there were limitations with the study, such as the use of an organic solvent for the vehicle, and the exclusion of skin bound residues, a dermal absorption of 17% was selected.

3.4.2 Non-occupational Exposure and Risk Assessment

Non-occupational risk assessment involves estimating risks to the general population, including youth and children, during or after pesticide application.

The United States Environmental Protection Agency (USEPA) has generated standard default assumptions for developing residential exposure assessments for both applicator and postapplication exposures when chemical- and/or site-specific field data are limited. These assumptions may be used in the absence of, or as a supplement to, chemical- and/or site-specific data and generally result in high-end estimates of exposure. These assumptions are outlined in the *Standard Operating Procedures for Residential Pesticide Exposure Assessment* (2012) (USEPA Residential SOPs).

The following sections from the USEPA Residential SOPs were used to assess residential exposure to d-phenothrin:

- Section 4: Gardens and Trees
- Section 7: Indoor Environments
- Section 8: Treated Pets

Hornet and wasp nests were considered under Sections 4 and 7, since the application methods were addressed in these sections.

3.4.2.1 Residential Applicator Exposure and Risk Assessment

A residential applicator would be an adult who purchased a domestic-class d-phenothrin product for use in and around the home. Residential applicators are assumed to be wearing shorts, short-sleeved shirts, shoes and socks. Homeowners have the potential for short-term exposure (1-30 days) when applying products containing d-phenothrin.

Based on typical use patterns, the major scenarios identified were:

- Applying dust formulations by shaker cans in indoor environments;
- Applying aerosol formulations to indoor environments;
- Applying aerosol formulations to outdoor gardens and trees, and hornet, wasp and yellow jacket nests;
- Applying liquid spot-on solutions to pet dogs.

Based on the short-term residential applicator assessment, the calculated MOEs are greater than the target MOE for all scenarios conducted for both dermal and inhalation exposures. As such, no mitigation measures are required for these scenarios. The results of the risk assessment are summarized in Appendix IV, Table 2.

3.4.2.2 Residential Postapplication Exposure and Risk Assessment

Postapplication exposure occurs when an individual is exposed through dermal, inhalation, and/or incidental oral (non-dietary ingestion) routes as a result of being in a residential environment that has been previously treated with a pesticide. The area could have been treated by a residential applicator using a domestic-class product or a commercial applicator hired to treat the residential area.

There is potential for intermittent short-term exposure to adults, youth (11 to < 16 years old), and children (6 to < 11 years old and 1 to < 2 years old) through contact with transferable residues following applications of d-phenothrin to indoor and outdoor environments and to pet dogs. Adults, youth and children have the potential for postapplication dermal exposure; children (1 to < 2 years old) also have the potential for incidental oral exposure. The highest application rate was used in the postapplication risk assessment for d-phenothrin.

Due to seasonality of most pests (for example, fleas) listed on the label, postapplication exposure is expected to be intermittent short-term (1-30 days). The following scenarios were assessed for short-term postapplication exposure for residential use of products containing d-phenothrin:

- Adults, youth and children (1 to < 2 years old) dermal and inhalation exposure resulting from activities indoors;
- Adults, youth and children (1 to < 2 years old) dermal exposure resulting from activities with treated pets;
- Adults, youth and children (6 to < 11 years old) dermal exposure resulting from activities in gardens, and around trees, and indoor plants;
- Incidental oral (hand-to-mouth and object-to-mouth) exposure to children (1 to < 2 years old) in indoor environments;
- Incidental oral (hand-to-mouth) exposure to children (1 to < 2 years old) from treated pets.

For bed bugs, there may be the potential for long-term exposure (> 180 days). The following scenarios were assessed for long-term postapplication exposure for residential use of products containing d-phenothrin for bed bugs:

- Adult, youth and children (1 to < 2 years old) dermal and inhalation exposure to surface directed spray applications of aerosol products used in indoor environments;
- Incidental oral (hand-to-mouth) exposure to children (1 to < 2 years old) from aerosol formulation products used in indoor environments.

It is assumed that individuals contact previously treated surfaces and pets on the same day the pesticide treatment is applied.

Multiple applications were considered for treatment of gardens and trees with d-phenothrin. The Outdoor Residential Exposure Task Force (ORETF) Use and Usage Survey states that the average number of applications to outdoor ornamental plants and shrubs for insect control is two per season. The average application interval between the first and second application was 2.75 weeks, 1.94 weeks between the second and third application, 1.91 weeks between the third and fourth application and 1.38 weeks between the fourth and fifth applications. Based on this information, two applications with a two-week interval was assumed for outdoor gardens and tree applications.

Multiple applications were not assessed for indoor and pet uses of d-phenothrin since exposure on the day of application (Day 0), without any dissipation was assumed for the entire duration of exposure.

Postapplication dermal exposure was calculated using activity-specific transfer coefficients, estimates for treated foliage, fur or surface residue, dislodgeable residue (residue transfer to skin) and exposure time. A transfer coefficient is a factor that relates exposure to dislodgeable residues and the amount of treated surface that a person contacts while performing activities in a given period (usually expressed in units of cm^2 per hour). It is specific to a particular population and activity (for example, adults gardening)

Postapplication dermal exposure to pesticides applied to stinging insect nests was considered to be minimal as there is a 24-hour period recommended before disposing of treated nests. Inhalation exposure to outdoor applications was considered to be minimal due to low vapour pressure and expected dilution in outdoor air. These assumptions are consistent with the USEPA Residential SOPs.

Based on the short-term and long-term residential postapplication assessment, the calculated MOEs are greater than the target MOEs for all scenarios conducted for dermal, inhalation and incidental oral exposures. As such, no mitigation measures are required for these scenarios. The results of the risk assessment are summarized in Appendix IV, Tables 3-16.

3.4.3 Occupational Exposure and Risk Assessment

Workers can be exposed to *d*-phenothrin through application of *d*-phenothrin and when entering a treated site.

3.4.3.1 Applicator Exposure and Risk Assessment

For commercial applications, there are potential exposures to applicators. Based on typical use patterns, the major scenarios identified were:

- Aerosol application in homes, non-food areas of restaurants, schools, nursing homes, warehouses, offices, apartments, hotels, motels, kennels and hospitals;
- Aerosol application to hornet, wasp and yellow jacket nests;
- Commercial application of spot-on treatments to dogs by veterinarians.

Commercial applicators may handle *d*-phenothrin for short or extended periods of time depending on the pest and use site. Applicators have the potential for intermittent short-term (1-30 days) to intermediate-term (30-180 days) exposure to *d*-phenothrin.

The following exposure scenarios were considered for commercial applicators:

A. Baseline Personal Protective Equipment – long pants, long-sleeved shirts and no gloves

Dermal and inhalation exposures were estimated using data from the Pesticide Handlers Exposure Database (PHED), Version 1.1. The PHED is a compilation of generic mixer, loader, and applicator passive dosimetry data with associated software that facilitates the generation of scenario-specific exposure estimates based on formulation type, application equipment, mix/load systems and level of PPE.

PHED aerosol data are representative of typical aerosol spray can applications of a pesticide with the exception of the use of aerosol cans with a stream-type nozzle (such as for wasp and hornet control). PHED data were generated by individuals applying a contact insecticide to the baseboards of kitchens. Hence, the exposure data in this scenario may underestimate upper body and inhalation exposure during the commercial use of stream-type nozzles, especially for application to higher cracks and crevices.

The calculated dermal and inhalation MOEs are well above the target MOE for all of the commercial applicator scenarios using baseline PPE. As such, no mitigation measures are required for these scenarios. The large MOEs for aerosol cans with stream-type nozzles indicates that even if exposure is underestimated, risks would not be of concern. The results of the risk assessment are summarized in Appendix IV, Table 1.

The insecticide d-phenothrin is available as a domestic-class spot-on product for pet treatment. Registrant survey data show that spot-on products containing d-phenothrin are not typically used in veterinary offices or by pet grooming services. However, it is assumed that this product may be used by commercial users such as veterinarians, veterinary technicians and groomers. Exposure is expected by the dermal route for veterinarians and other commercial users applying domestic spot-on products (such as groomers). There are no specific exposure data available for the commercial applicator spot-on scenario. Therefore, exposure was compared to the residential applicator in terms of amount of product handled, number of pets treated, personal protective equipment (PPE) worn, and margins of exposure. For commercial users, the extent of exposure is uncertain, however, these workers typically wear PPE when applying pet products, such as a laboratory coat/apron. The number of animals treated per day with the spot-on products by veterinarian offices is assumed to be eight animals per day based on registrant-provided information. It was assumed that applying pet products is only one of many tasks that would be done in a typical day, and it may not always be the same product being applied. Application of pet products may also be delegated to other staff within the veterinarian office. As MOEs for the residential spot-on applicator assessment were relatively large, it is expected that commercial exposures to spot-on applications would not result in risks of concern. As such, no mitigation measures are required for these scenarios. The results of the risk assessment are summarized in Appendix IV, Table 2.

5.6.2.2 Postapplication Worker Exposure and Risk Assessment

There is potential exposure to workers entering treated sites or handling treated pets.

Possible occupational postapplication worker scenarios include:

- Commercial applicator or pest control operator returning to treated sites for scouting;
- Workers in a treated commercial, industrial or institutional location;
- Workers in treated hotels and motels;
- Workers in treated boats, buses, ships or trains;
- Workers in treated nursing homes and hospitals;
- Workers in treated restaurants;
- Veterinarians or workers handling treated pets.

A specific assessment for postapplication workers was not conducted. It was assumed that risks to postapplication workers would be similar to or less than residential postapplication risks. No risks of concern were identified for residential postapplication scenarios. Given the degree by which the MOEs exceed the target MOEs for residential postapplication scenarios, this assumption is unlikely to underestimate occupational postapplication exposure.

5.7 Aggregate Risk Assessment

Aggregate exposure is the total exposure to a single pesticide that may occur from food, drinking water, residential, and other non-occupational sources, and from all known or plausible exposure routes (oral, dermal and inhalation).

5.7.1 Toxicology Endpoint Selection

Endpoints relevant to short-term exposures from route-specific studies were dissimilar (generalized toxicity in oral studies versus local effects in the inhalation studies) or non-existent (absence of effects at limit dose in dermal study). As such, only exposures from similar routes were aggregated (dietary and incidental oral).

The endpoint for long-term aggregate assessment by all routes of exposure is the same as that specified for the acceptable daily intake and long-term dermal and inhalation endpoint. As such, the exposures from each long-term route of exposure (dietary, dermal, inhalation, and incidental oral [children 1 - 2 years old]) were combined as appropriate and an aggregate MOE was calculated using the common endpoint and target MOE.

3.5.2 Residential and Non-occupational Aggregate Exposure and Risk Assessment

In an aggregate risk assessment, the combined potential risk associated with food, drinking water, and various residential exposure pathways is assessed. A major consideration is the likelihood of co-occurrences of exposure. Risks resulting from different exposure scenarios are combined when it is likely that they can occur simultaneously based on the use pattern and when the toxicological effects across different routes of exposure are the same. There were no common toxic adverse effects across different exposure routes for intermittent short-term exposure. As such, only exposure that could co-occur and have the same route of exposure were aggregated for short-term exposure scenarios (such as dietary and incidental oral).

For long-term exposure scenarios (bed bug applications) there is a common toxic adverse effect across dermal, inhalation, incidental oral (children 1 to < 2 years old), and dietary exposure. As such, postapplication exposures from dermal, inhalation, hand-to-mouth (children (1 to < 2 years old), and dietary exposure were aggregated for *d*-phenothrin.

For *d*-phenothrin, the following exposures were aggregated.

Table 1. Short-term Aggregated Exposures

Scenario	Population	Route	Aggregated Exposures	
Indoor Environments	Adult	Dermal	Applicator	Postapplication
		Inhalation	Applicator	Postapplication
	Children (1 < 2 yrs)	Oral	Hand-to-mouth	Dietary
Gardens and Trees	Adult	Dermal	Applicator	Postapplication
Treated Pets	Adult	Dermal	Applicator	Postapplication
	Children (1 < 2 yrs)	Oral	Hand-to-mouth	Dietary

Table 2. Long-term Aggregated Exposures

Scenario	Population	Aggregated Exposures			
Indoor Environments, postapplication exposure to bed bug treatments	Adult	Inhalation	Dermal	Dietary	—
	Youth	Inhalation	Dermal	Dietary	—
	Children (1< 2 yrs)	Inhalation	Dermal	Dietary	Hand-to-mouth

The resulting chronic dietary exposure estimates for aggregation with residential exposure are in Table 2, Appendix V.

Based on the short-term and long-term aggregate assessments, the calculated aggregate MOEs are greater than the target aggregate MOE for all scenarios conducted for dermal, inhalation, incidental oral and dietary exposures. As such, no mitigation measures are required for these scenarios. The results of the risk assessment are summarized in Appendix IV, Tables 17-19.

4.0 Impact on the Environment

4.1 Fate and Behaviour in the Environment

The insecticide d-phenothrin is used primarily in and around homes as a domestic insecticide. Based on this use pattern, environmental exposure is expected to be minimal.

Data on the fate and behaviour of d-phenothrin are summarized in Table 1, Appendix VII.

In soil, d-phenothrin transforms through biotransformation and is slightly persistent, with a half-life of 26 days. Results from United States field studies indicate that d-phenothrin quickly dissipates in soil and the transformation products were not detected below 15 cm.

In clear surface waters, d-phenothrin undergoes rapid phototransformation, with a half-life of 9 hours. In aquatic environments, d-phenothrin is also broken down via aerobic biotransformation with a half-life of 36 days. Hydrolysis is not an important route of transformation of d-phenothrin.

The insecticide d-phenothrin is practically insoluble and non-volatile. Although the log K_{ow} indicates that d-phenothrin has the potential to bioaccumulate, bioconcentration studies showed that bioconcentration factors (BCFs) were below the Toxic Substances Management Policy (TSMP) criteria cut-off of 5000. This, in addition to the minimal environmental exposure, indicates that bioaccumulation is not expected to be of concern. Based on the high K_{oc}, the results of terrestrial field dissipation studies and the limited use pattern, d-phenothrin is not expected to leach to groundwater.

4.2 Environmental Risk Characterization

The environmental risk assessment integrates the environmental exposure and ecotoxicology information to estimate the potential for adverse effects on non-target species. Available toxicity data for d-phenothrin is presented in Table 2, Appendix VII.

The use pattern indicates that the exposure of environmental compartments (soil, aquatic systems and food sources for birds and mammals) to d-phenothrin will be minimal. Therefore, expected environmental concentrations were not calculated and a quantitative risk assessment was not conducted.

4.2.1 Risks to Terrestrial Organisms

Due to the use pattern of d-phenothrin, the potential exposure of terrestrial non-target organisms is not expected to be significant. Therefore, the risk to terrestrial organisms is expected to be negligible.

4.2.2 Risks to Aquatic Organisms

Due to the limited outdoor use of d-phenothrin, exposure to aquatic habitats is expected to be minimal. Therefore, the risk to aquatic organisms is expected to be negligible.

5.0 Value

The insecticide d-phenothrin is registered in Canada for use on non-agricultural sites. It is currently registered for use in and around structures, on indoor and outdoor ornamental plants, as flea and tick control products, and human proximal sites for the control of numerous pests. These pests include bed bugs, hornets, wasps, yellow jackets, ants, black carpet beetles, crickets, fleas, sowbugs, spiders, brown dog ticks, cockroaches, centipedes, caterpillars, silverfish, earwigs, mealy bugs, saw-toothed grain beetles, confused flour beetles, rice weevils, fungus gnats, flies, mosquitoes, moths, leafhoppers, Japanese beetles and whiteflies.

The insecticide d-phenothrin contributes to resistance management by helping to delay the development of resistance when used in rotation with other insecticides with different modes of action.

6.0 Pest Control Product Policy Considerations

6.1 Toxic Substances Management Policy Considerations

The Toxic Substances Management Policy (TSMP) is a federal government policy developed to provide direction on the management of substances of concern that are released into the environment. The TSMP calls for the virtual elimination of Track 1 substances (those that meet all four criteria outlined in the policy: persistent [in air, soil, water and/or sediment], bioaccumulative, primarily a result of human activity and toxic as defined by the *Canadian Environmental Protection Act*).

During the review process, d-phenothrin and its transformation products were assessed in accordance with the PMRA Regulatory Directive DIR99-03[3] and evaluated against the Track 1 criteria. The PMRA has reached the following conclusions:

- The insecticide d-phenothrin does not meet Track 1 criteria, and is not considered a Track 1 substance. See Table 3, Appendix VII for comparison with Track 1 criteria.

- The insecticide d-phenothrin does not form any transformation products that meet all Track 1 criteria.

6.2 Formulants and Contaminants of Health or Environmental Concern

During the review process, contaminants in the technical and formulants and contaminants in the end-use products are compared against the *List of Pest Control Product Formulants and Contaminants of Health or Environmental Concern* maintained in the *Canada Gazette*[4]. The list is used as described in the PMRA Notice of Intent NOI2005-01[5] and is based on existing policies and regulations including: DIR99-03 and DIR2006-02[6] and taking into consideration the Ozone-depleting Substance Regulations, 1998, of the *Canadian Environmental Protection Act* (substances designated under the Montreal Protocol). The PMRA has reached the following conclusions:

- Technical grade d-phenothrin and the end-use products do not contain any formulants or contaminants of health or environmental concern identified in the *Canada Gazette*.

The use of formulants in registered pest control products is assessed on an ongoing basis through PMRA formulant initiatives and Regulatory Directive DIR2006-02.[4]

7.0 Incident Reports

Since 26 April 2007, registrants have been required by law to report incidents, including adverse effects to health and the environment, to the PMRA. Information on the reporting of incidents can be found on the Pesticides and Pest Management portion of Health Canada's website. As of 2 May 2014, a total of 25 human and 194 domestic animal incidents involving d-phenothrin have been reported to the PMRA.

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3 DIR99-03, *The Pest Management Regulatory Agency's Strategy for Implementing the Toxic Substances Management Policy.*

4 *Canada Gazette*, Part II, Volume 139, Number 24, SI/2005-114 (2005-11-30) pages 2641–2643: *List of Pest Control Product Formulants and Contaminants of Health or Environmental Concern* and in the order amending this list in the *Canada Gazette*, Part II, Volume 142, Number 13, SI/2008-67 (2008-06-25) pages 1611-1613. *Part 1 Formulants of Health or Environmental Concern, Part 2 Formulants of Health or Environmental Concern that are Allergens Known to Cause Anaphylactic-Type Reactions and Part 3 Contaminants of Health or Environmental Concern.*

5 NOI2005-01, *List of Pest Control Product Formulants and Contaminants of Health or Environmental Concern under the New Pest Control Products Act.*

6 DIR2006-02, PMRA Formulants Policy.

There was some degree of association between the symptoms and the reported exposure in 71% of the human incidents. Most of these incidents involved minor dermal, respiratory or general symptoms following exposure to a domestic-class product during application or through contact with a treated area. No significant human health concerns were identified based on the incident reporting data.

Most of the domestic animal incident reports involving d-phenothrin were related to use of a flea and tick control product. These incident reports will be included in the PMRA's ongoing evaluation of incident reports related to flea and tick control products. Very few domestic animal incidents were related to other types of products and were generally minor in severity.

These incident reports were considered in this evaluation and did not affect the risk assessment.

There were no environmental incidents involving d-phenothrin in the PMRA or USEPA databases.

8.0 Organisation for Economic Co-operation and Development Status of d-Phenothrin

Canada is part of the Organisation for Economic Co-operation and Development (OECD), which provides a forum in which governments can work together to share experiences and seek solutions to common problems.

As part of the re-evaluation of an active ingredient, the PMRA takes into consideration recent developments and new information on the status of an active ingredient in other jurisdictions, including OECD member countries.

The insecticide d-phenothrin is currently acceptable for use in other OECD countries, including the United States and Australia. As of 21 November 2014, no decision by an OECD member country to prohibit all uses of d-phenothrin for health or environmental reasons has been identified.

9.0 Proposed Re-evaluation Decision

The PMRA is proposing that products containing d-phenothrin for use and sale in Canada are acceptable for continued registration. Based on the evaluation of available scientific information, the health risks associated with d-phenothrin, under the current conditions of use, meet current standards. Therefore, mitigation measures are not required to further protect human health. However, labels require updating to ensure consistency in label statements and best practices, be consistent with the assumptions used in the health risk assessment and minimize unnecessary exposure. In addition, due to the limited outdoor use, the risk to terrestrial and aquatic organisms is expected to be minimal. Precautionary statements are proposed to further protect the environment.

The labels of Canadian end-use products must be amended to include the label statements listed in Appendix VIII. No additional data are being requested at this time.

10.0 Supporting Documentation

PMRA documents, such as Regulatory Directive DIR2012-02, *Re-evaluation Program Cyclical Re-evaluation*, and DACO tables (datacode tables) can be found on the Pesticides and Pest Management portion of Health Canada's website. PMRA documents are also available through the Pest Management Information Service. Phone: 1-800-267-6315 within Canada or 1-613-736-3799 outside Canada (long distance charges apply); fax: 613-736-3798; e-mail: pmra.infoserv@hc-sc.gc.ca.

The federal Toxic Substances Management Policy is available through the Environment Canada website.

List of Abbreviations

ADI	acceptable daily intake
a.i.	active ingredient
A/G	albumin/globulin ratio
ALB	albumin
ALP	alkaline phosphatase
AR	application rate
ARfD	acute reference dose
BAF	bioaccumulation factor
BCF	bioconcentration factor
BUN	blood urea nitrogen
bw	body weight
CAF	composite assessment factor
CAS	Chemical Abstracts Service
CEPA	*Canadian Environmental Protection Act*
cm	centimetres
DA	dermal absorption
DACO	data code
DNA	deoxyribonucleic acid
DU	dust or powder formulation
et al.	and others
F_1	first generation
F_2	second generation
fc	food consumption
g	gram(s)
g a.i.	grams of active ingredient
GD	gestation day
ha	hectare(s)
HDT	highest dose tested
Hct	hematocrit
Hgb	hemoglobin
ICR mice	importing control region mice
kg	kilogram(s)
K_{oc}	organic-carbon partition coefficient
K_{ow}	octanol-water partition coefficient
L	litre(s)
LC_{50}	lethal concentration to 50%
LD_{50}	lethal dose to 50%
LDH	lactate dehydrogenase
LOAEL	lowest observed adverse effect level
m	metre(s)
m^3	metres cubed
MAS	maximum average score for 24, 48 and 72 hours
mg	milligram(s)
MIS	maximum irritation score
MOE	margin of exposure

MIS	maximum tolerated dose
NADPH	nicotinamide adenine dinucleotide phosphate
NOEC	no observed effect concentration
NOAEL	no observed adverse effect level
OECD	Organisation for Economic Co-operation and Development
ORETF	Outdoor Residential Exposure Task Force
P	parental generation
PCPA	*Pest Control Products Act*
PHED	Pesticide Handlers Exposure Database
PPE	personal protective equipment
PMRA	Pest Management Regulatory Agency
ppm	parts per million
RBC	red blood cells
SN	solution
SOP	standard operating procedure
µg	microgram(s)
USEPA	United States Environmental Protection Agency
VUI	verified use information
wt(s)	weight(s)
WBC	white blood cells

Appendix I Registered d-Phenothrin Products as of 11 June 2013[1]

Registration Number	Marketing Class	Registrant	Product Name	Formulation Type	Guarantee
30781	Domestic	Aerokure International Inc.	Insect Stop Bed Bug Killer	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
29422		Agrium Advanced Technologies RP INC.	Pro Aerosol Kills Bedbugs	Pressurized product	d-phenothrin 0.12%; n-octyl bicycloheptene dicarboximide 1.00%; piperonyl butoxide 0.980%; prallethrin 0.025%
30070		Alts Packaging Systems Inc.	Kablamo Bed Bug Killer	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
30701		Auza Pro Solutions Inc.	Zone Guard, Bed Bug Killer	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
30704			Zone Guard, Pro Ant, Roach & Crawling Insect Blaster		d-phenothrin 0.50%; n-octyl bicycloheptene dicarboximide 1.00%; imiprothrin 0.40%
30706			Zone Guard, Wasp & Hornet Blaster		d-phenothrin 0.125%; tetramethrin 0.200%
30069		Brodi Specialty Products Ltd.	Brodi Bed Bug Killer	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
30578		Business Helpers' Depot Inc.	Fight Back Insecticide M-3-2	Pressurized product	d-phenothrin 0.125%; tetramethrin 0.20%
30425	Commercial	Cantol Corp.	Wasp & Hornet Spray	Pressurized product	d-phenothrin 0.125%; tetramethrin 0.20%
30689	Domestic	Consol International Inc.	Bed Bug Fix	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
27874		CRC Industries Inc.	CRC Bug Blast Wasp And Hornet Killer	Pressurized product	d-phenothrin 0.125%; tetramethrin 0.20%
30285		DCG Vision Marketing & Sales International Ltd.	Power Shot Bed Bug Killer	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
30306		Direct Line Sales & Supplies Corp.	Onguard T 20	Pressurized product	d-phenothrin 0.125%; tetramethrin 0.20%
25226		Hartz Canada Inc.	Hartz Ultraguard Flea & Tick Carpet Powder	Dust or powder	d-phenothrin 0.50%; n-octyl bicycloheptene dicarboximide 1.18%

Registration Number	Marketing Claim	Registrant	Product Name	Formulation Type	Guarantees
30729			Hartz Ultraguard Pro Flea & Tick Treatment for Dogs and Puppies Weighing 6 kg to 14 kg	Solution	deltamethrin 65.76% methoprene 2.30%
30729			Hartz Ultraguard Pro Flea & Tick Treatment for Dogs and Puppies Weighing 2.5 kg to 6 kg		
30730			Hartz Ultraguard Pro Flea & Tick Treatment for Dogs and Puppies Weighing 14 kg to 28 kg		
30731	Domestic	Hartz Canada Inc.	Hartz Ultraguard Pro Flea & Tick Treatment for Dogs and Puppies Weighing Greater than 28 kg	Solution	deltamethrin 65.76% methoprene 2.30%
30732			Hartz Ultraguard Flea & Tick Treatment for Dogs and Puppies Weighing 2.5 kg to 6 kg		deltamethrin 65.76%
30733			Hartz Ultraguard Flea & Tick Treatment for Dogs and Puppies Weighing 6 kg to 14 kg		
30734			Hartz Ultraguard Flea & Tick Treatment for Dogs and Puppies Weighing 14 kg to 28 kg		
30735			Hartz Ultraguard Flea & Tick Treatment for Dogs and Puppies Weighing Greater than 28 kg		
24959		K-G Spray-Pak Inc.	K-G Home & Garden Insect Killer VI	Pressurized product	deltamethrin 0.20% cyfluthrin 0.20%
24960			K-G Flying Insect Killer III		deltamethrin 0.15% cyfluthrin 0.25%
24783			K-G Wasp & Hornet Killer		deltamethrin 0.125% cyfluthrin 0.20%
24946			Better Than Bad Bug Killer		deltamethrin 0.20% cyfluthrin 0.20%
30273			Better Than Wasp & Hornet Killer I		deltamethrin 0.125% cyfluthrin 0.002%

Registration Number	Marketing Class	Registrant	Product Name	Formulation Type	Guarantee
24825	Domestic	KUUS Inc.	Knock Down Flying Insect Killer I	Pressurized product	d-phenothrin 0.15%; tetramethrin 0.25%
[illegible]			Knock Down Bed Bug Killer I		d-phenothrin 0.20% tetramethrin 0.20%
[illegible]			Protex Bed Bug Killer .20% d-Phenothrin		
[illegible]			Knockdown Total Home Multi Flying & Crawling Insect Killer		
[illegible]			Knock Down Hornet & Wasp Blaster Spray Killer		d-phenothrin 0.125%; tetramethrin 0.20%
[illegible]		La Coop du Québec	Eliminator Plus Wasp & Hornet Killer Insecticide I	Pressurized product	d-phenothrin 0.125%; tetramethrin 0.002%
[illegible]		Les Marques Metro S.E.N.C.	Selection House & Home Insect Killer	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
29947		Les Produits de Contrôle Supérieur Inc. / Superior Control Products Inc.	Pro Maxx Bed Bug Destroyer	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
[illegible]			Super Hunter Wasp & Hornet Killer Insecticide I		d-phenothrin 0.125%; tetramethrin 0.002%
[illegible]	Domestic	Lloyds Laboratories	Wasp & Hornet Blaster	Pressurized product	d-phenothrin 0.125%; tetramethrin 0.20%
[illegible]	Commercial	McLaughlin Gormley King Company	Bedlam® Insecticide	Pressurized product	d-phenothrin 0.40%; n-octyl bicycloheptene dicarboximide 1.60%
24823	Domestic		Multicide Crawling Insect Killer		d-phenothrin 0.20%; tetramethrin 0.20%
24824			Multicide House & Garden Insect Killer		
25491			Nylar Pressurized Spray 2618		d-phenothrin 0.30%; pyriproxifen 0.015%; tetramethrin 0.40%
[illegible]			Multicide Wasp & Hornet Killer 2695		d-phenothrin 0.125%; tetramethrin 0.20%
27185			Multicide Pressurized Roach Spray 27341		d-phenothrin 0.50%; n-octyl bicycloheptene dicarboximide 1.0%; imiprothrin 0.400%

Registration Number	Marketing Class	Registrant	Product Name	Formulation Type	Guarantee
27404			ETOC Pressurized Spray 2594		d-phenothrin 0.12%; n-octyl bicycloheptene dicarboximide 1.0%; piperonyl butoxide 0.58%; prallethrin 0.025%
30745			Evercide® Wasp & Hornet Killer 20861		d-phenothrin 0.20%; tetramethrin 0.20%
24818	Manufacturing concentrate		Multicide Sumithrin 90% Concentrate	Solution	d-phenothrin 90.0%
24819	Manufacturing concentrate	McLaughlin Gormley King Company	Multicide Intermediate 2084	Solution	d-phenothrin 7.17%; tetramethrin 12.50%
24820			Multicide Intermediate 2086	Solution	d-phenothrin 8.90%; tetramethrin 8.90%
24829			Multicide Intermediate 2317	Emulsifiable concentrate	d-phenothrin 9.23%; tetramethrin 12.30%
26997			Multicide Intermediate 2660	Solution	d-phenothrin 6.25%; tetramethrin 10%
27384			Multicide Intermediate 2734	Emulsifiable concentrate	d-phenothrin 10%; imiprothrin 8%; n-octyl bicycloheptene dicarboximide 20%
27390			ETOC Concentrate 2593	Emulsifiable concentrate	d-phenothrin 3.43%; n-octyl bicycloheptene dicarboximide 28.58%; piperonyl butoxide 16.46%; prallethrin 0.72%;
30074			Multicide® Intermediate 2791	Solution	d-phenothrin 8%; n-octyl bicycloheptene dicarboximide 32%
30033	Domestic	Natures' Innovation Inc.	Bed Bug Patrol Bed Bug Killer	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
30287	Domestic	NCH Canada Inc.	X-Pire	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
30126	Domestic	Night Bug Enr.	Night Bugs Bed Bug Killer	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
30303	Domestic	Novella Brands Inc.	Blaze Wasp & Hornet Killer	Pressurized product	d-phenothrin 0.125%; tetramethrin 0.20%
30994			Blaze Professional Wasp & Hornet Killer		

Registration Number	Marketing Class	Registrant	Product Name	Formulation Type	Guarantee
27498	Domestic	Professional Pet Products Inc.	Cycleblock Home & Carpet Spray	Pressurized product	d-phenothrin 0.30%; pyriproxifen 0.015%; tetramethrin 0.40%
26502	Domestic	Rolf C. Hagen Inc.	Sergeant's Pretect Household Flea Spray	Pressurized product	d-phenothrin 0.30%; pyriproxifen 0.015%; tetramethrin 0.40%
25504	Domestic	S.C. Johnson and Son Ltd.	Raid Flying Insect Killer 2	Pressurized product	d-phenothrin 0.150%; piperonyl butoxide 0.48%; d-cis, trans allethrin 0.15%
26932			Raid Max House & Garden Multi-Bug Killer		d-phenothrin 0.20%; tetramethrin 0.20%
29696			Raid Max Crawling Insect Bug Killer 2		
29697			Raid Ant, Roach & Earwig Bug Killer 18		
29699			Raid Spider Blaster Bug Killer 3		
29776			Raid Wasp & Hornet Bug Killer 7		d-phenothrin 0.125%; tetramethrin 0.20%
30744			Raid® Outdoor Ant Nest Destroyer 2		d-phenothrin 0.20%; tetramethrin 0.20%
30746			Raid® Max Wasp & Hornet Foam Bug Killer 2		
30557	Domestic	Scotts Canada Ltd.	Ortho® Home Defense® Max, Hornet & Wasp Eliminator Spray	Pressurized product	d-phenothrin 0.125%; tetramethrin 0.20%
29204	Domestic	Shoppers Drug Mart/Pharmaprix	Life Brand House & Home Insect Killer	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
28380	Commercial	State Industrial Products	Zero In Was Wasp & Hornet Killer	Pressurized product	d-phenothrin 0.125%; tetramethrin 0.20%
24817	TGAI	Sumitomo Chemical Company, Limited	Sumithrin Technical Grade	Solution	d-phenothrin 96.6%

Registration Number	Marketing Class	Registrant	Product Name	Formulation Type	Guarantee
26179	Domestic	Sure-Gro IP INC.	C-I-L Flea Killer Surface Spray	Pressurized product	d-phenothrin 0.30%; Pyriproxifen 0.015%; tetramethrin 0.40%
26208			C-I-L House and Garden Insect Killer 2		d-phenothrin 0.20%; tetramethrin 0.20%
26836			Schultz House Plant Insect Spray		
28788			Wilson One Shot House & Garden Insect Killer		
29423			Schultz Houseplant Insect Spray		
29426			Wilson Oneshot House and Indoor Garden Insect Killer		
29555			C-I-L House and Indoor Garden Insect Killer		
30158			Green Earth Homecare Bed Bug Travel Spray		
30191			C-I-L Wasp & Hornet Long Shot		d-phenothrin 0.125%; tetramethrin 0.20%
30192			Wilson Oneshot Wasp & Hornet Long Shot		
30432			Green Earth Homecare Flying & Crawling Insect Killer (1)		d-phenothrin 0.20%; tetramethrin 0.20%
29729	Domestic	Ultrasol Industries Ltd.	Doktor Doom Sleep Tight – Fabrics and Upholstery Professional Strength Bed Bug Killer	Pressurized product	d-phenothrin 0.12%; n-octyl bicycloheptene dicarboximide 1.0% ; piperonyl butoxide 0.58%; prallethrin 0.025%
30777			Doktor Doom Wasp & Hornet Nest Annihilator	Pressurized product	d-phenothrin 0.125%; tetramethrin 0.20%
30975	Domestic	Ur-CAN Inc.	Eco-Guard Wasp & Hornet Blaster	Pressurized product	d-phenothrin 0.125%; tetramethrin 0.20%
28841	Domestic	Wal-Mart Canada Inc.	Great Value® House and Home Insect Killer	Pressurized product	d-phenothrin 0.20%; tetramethrin 0.20%
30879	Domestic	Woodstream Canada Corporation	Terro® Wasp & Hornet Killer	Pressurized product	d-phenothrin 0.125%; tetramethrin 0.20%

[1] Discontinued products and products with a submission for discontinuation have not been included.

Appendix IIa Commercial Class Uses of d-Phenothrin Registered in Canada as of 11 June 2013[1]

Site(s)	Pest(s)	Formulation Type	Application Methods and Equipment	Maximum Single Application Rate	Supported Use?	Comments
Use Site Category 20: Structures						
Indoor use only in homes and non-food areas of restaurants, schools, nursing homes, warehouses, offices, apartments, hotels, motels, kennels and hospitals.	Bed bugs and bed bug eggs	Pressurized product	Crack and crevice (pressurized spray can)	2.4 g a.i./can	Yes	Maximum container size: 600 g
Indoor: carpets, box springs, walls, furniture, floor and floor coverings, closets and window-treatment hardware.			Spot spray (pressurized spray can)			
Use Site Category 26: Human Skin, Clothing and Proximal Sites						
Indoor: mattresses	Bed bugs and bed bug eggs	Pressurized product	Crack and crevice (pressurized spray can)	2.4 g a.i./can	Yes	Maximum container size: 600 g
			Spot spray (pressurized spray can)			
Use Site Category 20 and 33: Structures and Residential Outdoors						
Hornet, wasp and yellow jacket nests	Hornets, wasps and yellow jackets	Pressurized product	Wasp and hornet spray (pressurized spray can)	0.81 g a.i./can	Yes	Maximum container size: 650 g

[1] Discontinued products and products with a submission for discontinuation have not been included.

Appendix IIb Domestic Class Uses of d-Phenothrin Registered in Canada as of 11 June 2013[1]

Site(s)	Pest(s)	Formulation Type	Application Methods and Equipment	Maximum Single Application Rate	Supported Use?	Comments
Use Site Category 20: Structures						
Apartments, automobiles, boats, campers, homes, pet sleeping areas, recreation vehicles and storage areas	Ants, bed bugs, black carpet beetles (adults), crickets, fleas (adults), sowbugs, spiders and brown dog ticks	Pressurized product	Crack and crevice (pressurized spray can)	0.077 g a.i./m^2	Yes	Maximum container size: 207 g
			Spot spray (pressurized spray can)			
Indoor: carpets and upholstery	Fleas and ticks	Dust or Powder	Powder-area treatment (shaker can)	0.227 g a.i./m^2	Yes	Maximum container size: 500 g
Indoor (homes, apartments and kitchen)/Outdoor	Ants, cockroaches (American, German and Oriental), crickets, sowbugs, spiders, brown dog ticks, fleas, carpet beetles, centipedes, caterpillars, crickets, silverfish, earwigs, mealybugs, sawtoothed grain beetles, confused flour beetles, rice weevils, flies, mosquitos, bees, wasps, hornets, moths, fungus gnats and whiteflies	Pressurized product	Crack and crevice (pressurized spray)	3.10 g a.i./can	Yes	Maximum container size: 11.
			Spot spray (pressurized spray can)			
Use Site Category 24: Companion Animals						
Dogs (2.5 to 6 kg)	Fleas (adults, eggs and larvae), mosquitoes and ticks	Solution	Spot-on application (applicator tube)	0.576 g a.i./animal	Yes	Typical interval between applications: 1 month
Dogs (6 to 14 kg)				1.153 g a.i./animal		
Dogs (14 to 28 kg)				3.640 g a.i./animal		
Dogs over 28 kg				5.233 g a.i./animal		
Use Site Category 26: Human Skin, Clothing and Proximal Sites						
Indoor: mattress	Bed bugs	Pressurized product	Spot spray (pressurized spray can)	1.2 g a.i./can	Yes	Maximum container size: 600 g

Site(s)	Pest(s)	Formulation Type	Application Methods and Equipment	Maximum single application Rate	Supported Use?	Comments
Use Site Category 27: Ornamentals Outdoor						
Outdoor flower and ornamental plants	Aphids, Japanese beetles, mite, caterpillars, leafhoppers, spidermites, whiteflies, mealybugs and cutworm/cut caterpillars	Pressurized product	Spot spray (pressurized spray can)	0.2 g a.i./can	Yes	Maximum container size 600 g
Use Site Category 29: Outdoor Plants and Plantscape						
Houseplants	Mite, aphids, caterpillars, leafhoppers, spiders, whiteflies, mealybugs and spider mites	Pressurized product	Spot spray (pressurized spray can)	0.2 g a.i./can	Yes	Maximum container size 600 g
Use Site Category 29 and 35: Structures and Residential Outdoors						
Hornet nests, wasp nests and yellow jacket nests	Hornets, wasps and yellow jackets	Pressurized product	Contact spray (pressurized spray can)	0.2 g a.i./can	Yes	Maximum container size 600 g

Discontinued products and product with a submission for discontinuation have not been included

Appendix III Toxicology Assessment for d-Phenothrin

Table 1 Toxicity Profile of d-Phenothrin

Effects are known or assumed to occur in both sexes unless otherwise noted; in such cases, sex-specific effects are separated by semicolons. Organ weight effects reflect both absolute organ weights and relative organ to body weights unless otherwise noted.

Study Type / Animal / PMRA No.	Study Results
Pharmacokinetic Study Sprague-Dawley rats PMRA Nos. 1216239, 2221847 and 1874093	**Absorption** The insecticidce d-phenothrin was rapidly absorbed after both single (4 or 200 mg/kg bw) and repeated (4 mg/kg bw/day) oral doses. **Distribution** Tissue residues were low with radioactivity identified primarily in the fat for both isomers. Fat levels of the trans-isomer were 2% to 10% lower than those of the cis-isomer. Tissue levels were higher following repeated doses than following a single dose with the concentration of radioactivity in the brain, liver, kidney and blood reaching maximum levels three hours post-dosing. At 24 hours post-dosing, the concentration of radioactivity in these tissues decreased to one-tenth to one-twentieth of the maximum concentration that was noted three hours post-dosing. Compared to the liver and kidneys, the brain contained a small amount of radioactivity. **Metabolism** Most urinary metabolites were derived following ester cleavage. The major metabolites identified in the urine and feces were 3-(4'-hydroxy)phenoxybenzoic acid and 3-phenoxybenzoic acid. A major metabolite identified in the feces was intact d-phenothrin, which was considered to be unabsorbed material. The metabolite pattern appeared to be the same regardless of the dose regimen. **Excretion** Following either single or repeated doses of both cis- and trans-isomers, excretion of administered radioactivity was virtually complete within 7 days (96% to 100% recovered). After single doses of either isomer, the primary route of excretion was the feces (range of 55.9% to 86.6%) with fecal excretion higher for the cis-isomer. Urinary excretion after single doses of either isomer ranged from 10.8% to 40.1%. After repeated doses, the fecal route was still the predominant route for the cis-isomer (feces: 71.7% to 72.9%) while the urinary excretion was the predominant route of elimination for the trans-isomer (urine: 70.3% to 74.9%). There was no detectable radioactivity in expired carbon dioxide. **Whole-body Autoradiography** The radioactivity was rapidly distributed into tissues and organs following dosing. The greatest concentration of radioactivity in the tissues was found three hours post-dosing. At 24 hours post-dosing, most of the radioactivity disappeared from the tissues. No significant sex differences were identified in distribution, metabolism or excretion.

Study Type / Animal / PMRA No.	Study Results
Acute Oral Toxicity Sprague-Dawley rats PMRA Nos. 2222322 and 1874095	LD_{50} > 5000 mg/kg bw Low toxicity
Acute Dermal Toxicity Sprague-Dawley rats PMRA Nos. 1157417 and 1874095	LD_{50} > 2000 mg/kg bw Low toxicity
Acute Inhalation Toxicity ICR mice PMRA No. 2126786	LC_{50} > 1.18 mg/L Slight toxicity
Acute Inhalation Toxicity Sprague-Dawley rats PMRA Nos. 2126819, 2126786 and 1874095	LC_{50} > 1.18 to 2.1 mg/L Low to slight toxicity
Eye Irritation New Zealand White rabbits PMRA Nos. 1142183 and 1874095	MAS = 0.33 MIS = 1, at 1 and 24 hr Minimally irritating
Dermal Irritation New Zealand White rabbits PMRA Nos. 1142183 and 1874095	MAS = 0 MIS = 0 Non-irritating
Dermal Sensitization (Maximization test) Hartley guinea pigs PMRA Nos. 1142184 and 1874095	Non-sensitizer
5-Week Range-Finding Dietary Toxicity Study B6C3F1 mice PMRA Nos. 1233959 and 1874093	No NOAEL established (range-finding). Effects observed at ≥ 230 mg/kg bw/day included the liver and the kidneys.
13-Week Dietary Toxicity Study F344 rats	NOAEL = 70/75 mg/kg bw/day ≥ 216/227 mg/kg bw/day: ↓ total cholesterol; ↑ ALP (5 wks) and A/G ratio, ↓ absolute spleen wt (♂); ↑ total plasma protein, ALB and liver wts (♀)

Study Type / Animal / PMRA No.	Study Results
PMRA Nos. 1142584, 1233961 and 1874093	706/714 mg/kg bw/day: ↓ fc and ↑ lymphocytes; ↓ bwg, ↑ hgb, WBC, ALP, LDH and ALB (11 wks), ↑ liver wts (♂); perigenital wetness and staining, ↑ WBC (5 wks), lymphocytes (5 wks) and A/G ratio, ↓ hgb (5 wks) and glucose (11 wks), ↓ thymus wt, μ uterine hydrometra (♀)
6-Month Dietary Toxicity Study Sprague-Dawley rats PMRA No. 1143141	NOAEL = 150 mg/kg bw/day 500 mg/kg bw/day: ↓ bwg (3 mths), ↑ ALB, A/G ratio and BUN, ↓ sodium, ↑ kidney, liver and adrenal wts; ↑ absolute liver wts (3 mths), ↓ RBC, hgb and hct, ↑ BUN (3 mths) and serum cholesterol, dilatation of the retinal vessels (♂); ↓ bw (3 mths), ↓ water intake and serum cholinesterase activity, ↑ relative thyroid and kidney (3 mths) wts, ↑ ALP (3 mths), ↑ lymphocytes (3 mths) and ↓ neutrophils (3 mths), ovarian cysts filled w/fluid (♀)
5-Day Range-finding Oral Toxicity Study New Zealand White rabbits PMRA No. 1227040	No NOAEL established (range-finding). Effects noted at ≥ 250 mg/kg bw/day included clinical signs.
26-Week Dietary Toxicity Study Beagle dogs PMRA Nos. 2126801, 2126802, 2126804, 2126806 and 1874093	NOAEL = 32/33 mg/kg bw/day (HDT)
52-Week Dietary Toxicity Study Beagle dogs PMRA Nos. 1216240, 1874095 and 1874093	NOAEL = 8.2/7.1 mg/kg bw/day ≥ 28/27 mg/kg bw/day: ↓ ALB and A/G ratio, pituitary microcysts; focal degeneration of adrenal cortex with mononuclear cell infiltration of adrenal glands, diffuse hepatocellular enlargement, focal mononuclear infiltration of epididymides (♂) 80 mg/kg bw/day: emesis, ↓ RBC, hgb and hct, ↑ ALP, ↑ liver wts, diffuse hepatocellular enlargement, focal degeneration of adrenal cortex with mononuclear cell infiltration; focal mononuclear infiltration of epididymides (♂); pituitary microcysts (♀)
21-Day Dermal Toxicity Study CD rats PMRA Nos. 1157418, 1874095 and 1874093	NOAEL = 1000 mg/kg bw/day No systemic effects observed. Desquamation of the skin observed in ♀ at ≥ 100 mg/kg bw/day and in ♂ at 1000 mg/kg bw/day.
4-Week Inhalation Toxicity Study ICR mice PMRA No. 2126786	NOAEL = 0.06 mg/L 0.21 mg/L: ↑ liver wts; ↑ hgb, hct and sedimentation values, ↑ pituitary and absolute adrenal wts (♂); ↓ hgb, hct and sedimentation values, ↓ pituitary and adrenal wts (♀) Depilation around the nose was observed at all dose levels, which spread over the whole body. Depilation was not noted following two weeks recovery.
4-Week Inhalation Toxicity Study	NOAEL = 0.063 mg/L 0.21 mg/L: ↑ RBC and hgb, ↓ sedimentation values; ↓ bwg, ↓ hct, ↑ pituitary,

Study Type / Animal / PMRA No.	Study Results
Sprague-Dawley rats PMRA No. 2126786	Adrenal and thyroid wts (♂); ↓ pituitary, adrenal, thyroid and ovarian wts (♀) No treatment-related effects were noted during the three-week recovery period.
90-Day Inhalation Toxicity Study Sprague-Dawley rats PMRA Nos. 1157419, 1874095 and 1874093	NOAEL = 0.104 mg/L ≥ 0.291 mg/L: eosinophilic inclusions in the olfactory epithelial cells of the nasal turbinates 1.066 mg/L: clinical signs of toxicity (poorly groomed fur, dirty tails and a reduced response to a sharp knock on the chamber door), ↑ liver wts, ↓ thrombotest time; ↑ absolute kidney wts, cortical vacuolation of adrenals (♂); ↑ staining of urogenital region, ↑ ALP and eosinophils, ↑ absolute thyroid and adrenal wts, centrilobular hepatocyte enlargement and follicular thyroid cell enlargement (♀)
104-Week Chronic Toxicity/ Carcinogenicity Study B6C3F1 mice PMRA Nos. 1210991, 1211104, 1236486, 1233957, 1874093 and 1874093	NOAEL = 45 mg/kg bw/day **Main Study** ≥ 150 mg/kg bw/day: ↓ bwg, ↑ liver wts, mild hepatomegaly (12 mths onward), clear cell foci/areas and nodular hyperplasia of the liver (♂) 450 mg/kg bw/day: ↓ relative kidney wts, ↓ WBC and lymphocytes, ↓ incidence of nephrocalcinosis (♂); ↓ bw and bwg (first 60 wks), ↑ liver and kidney wts, ↓ LDH, mild hepatomegaly and nodular hyperplasia of the liver (♀) **Interim Sacrifice** ≥ 150 mg/kg bw/day: mild hepatomegaly (♂); ↓ LDH (♀) 450 mg/kg bw/day: hepatocyte hypertrophy w/eosinophilia, ↑ lung wts and congestion of lungs; ↓ bwg (up to 60 wks), ↑ platelets (♂) Increased combined incidence of hepatocellular adenomas and carcinomas in ♀ (not statistically significant) only marginally exceeded historical control range. Equivocal evidence of carcinogenicity in females.
105/118-Week Dietary Chronic Toxicity/ Carcinogenicity Study Fisher 344 rats PMRA Nos. 1211105, 1211106, 1149486, 1236487 and 1874093	NOAEL = 47/56 mg/kg bw/day **Main Study** 141/168 mg/kg bw/day: ↓ ALT (♂: 49 & 77 wks; ♀: 25, 49 & 77 wks); ↑ relative liver wts, dilatation of sinuses in mesenteric lymph nodes and hepatocytic hypertrophy (♂); ↓ bwg (up to 76 wks) and AST (25, 49 & 77 wks) (♀) **Interim Sacrifice** 141/168 mg/kg bw/day: ↑ relative liver wts; ↓ bwg (♀) Increased incidence of adenomas and carcinomas of the preputial gland; however this finding was not repeated in a second study in rats at significantly greater dose levels (PMRA Nos. 1166306, 1166307, 1166308, 1874093 and 1874093).
104-Week Chronic Toxicity/ Carcinogenicity Study Fisher 344 rats PMRA Nos. 1166306, 1166307, 1166308, 1874093 and 1874093	NOAEL = 51/63 mg/kg bw/day **Main Study** ≥ 531/653 mg/kg bw/day: clinical signs of toxicity (hunched posture, urinary staining and a thin build), ↓ bw, bwg and fc, ↑ liver wts, ↓ absolute heart wts; ↑ ALP, ↓ LDH and urine volume (♂); ↑ platelets, LAP, ALB and A/G ratio, ↓ phospholipids, total cholesterol and fibrinogen, ↑ brain wts, panacinar hepatocytic hypertrophy, posterior capsular opacity, pale areas in the lungs (♀)

Study Type / Animal / PMRA No.	Study Results
	1,116/1,351 mg/kg bw/day: ↓ MCV and MCH, panacinar hepatocytic hypertrophy; clinical signs of toxicity (pallor, pale eyes, piloerection, ↓ activity and ↓ body temperature), ↑ glucose, phospholipids, total cholesterol, A/G ratio, clotting times and specific gravity of urine, ↓ calcium and fibrinogen, lymphocytic cell infiltration of the harderian gland and periacinar coagulative necrosis (♂); ↑ AST, γ-glutamyl transferase and potassium, ↓ PCV, hgb, CPK and LDH, ↑ adrenal wts, abnormal shaped kidneys, luminal dilatation of the uterus (♀) **Interim Sacrifice** ≥ 51/63 mg/kg bw/day: ↑ LAP, ALB and A/G ratio, ↓ PCV, fibrinogen, CPK, LDH, triglycerides, phospholipids and total cholesterol (♀) ≥ 531/653 mg/kg bw/day: clinical signs of toxicity (hunched posture, urinary staining and a thin build), ↓ bwg, fc and fe, ↓ fibrinogen, MCV, CPK, LDH and triglycerides, ↑ relative adrenal and liver wts, ↓ absolute heart wts, diffuse hypertrophic hepatocytes; ↑ ALB, potassium and specific gravity of urine, ↓ fibrinogen, MCH, LDH, glucose, triglycerides and urine pH, ↑ relative brain wts (♂); ↑ platelets, ALP, γ-glutamyl transferase, potassium and specific gravity of urine, ↓ MCH, RBC, hgb and urine pH, ↑ brain and absolute liver wts (♀) 1116/1351 mg/kg bw/day: diffuse hypertrophic hepatocytes; clinical signs of toxicity (pallor, ↓ body temperature, pale eyes, piloerection and ↓ activity), ↑ prothrombin time, activated partial thromboplastin time, activated partial thrombin time, ALP and A/G ratio, ↓ platelets, phospholipids, total cholesterol and calcium, ↑ absolute liver and relative heart wts, ↓ spleen wts (♂) Increased incidence of hepatocellular adenomas and carcinomas at the high dose in both sexes. Increased incidence of uterine adenomas and adenocarcinomas in high dose ♀. Evidence of carcinogenicity at doses greater than the MTD.
2-Generation Dietary Reproduction Toxicity Study Sprague-Dawley rats PMRA Nos. 1166309, 1874095 and 1874093	**Parental Toxicity** NOAEL = 59/70 mg/kg bw/day 177/208 mg/kg bw/day: ↓ bw (F_1) and bwg (F_0 and F_1), ↓ fc (F_1: GDs 15-20), bile duct proliferation (F_1); ↓ absolute testicular wts (F_1) (♂); ↑ liver wts (F_0), hepatocellular hypertrophy (F_0) (♀) 582/664 mg/kg bw/day: ↓ bw (F_1) and bwg (F_0 and F_1), ↑ liver (F_0 and F_1) and relative brain wts (F_1), ↓ absolute brain wts (F_1), hepatocytic hypertrophy (F_1) and bile duct proliferation (F_1); ↓ fc and fe (F_0), ↓ absolute testicular and epididymal wts (F_1), ↑ relative testicular and seminal vesicle wts (F_1) (♂); ↓ fc (F_1: GDs 15-20), ↓ ovary, non-gravid uterine and absolute pituitary wts (F_0 and F_1), ↑ absolute liver wt (F_1), hepatocellular hypertrophy (F_0) and bile duct proliferation (F_0) (♀) **Reproductive Toxicity** NOAEL = 59/70 mg/kg bw/day 177/208 mg/kg bw/day: ↑ number of stillborn pups (F_1) 582/664 mg/kg bw/day: ↓ mean number of corpora lutea (F_0 and F_1) and an ↑ number of stillborn pups (F_0 and F_1) **Offspring Toxicity** LOAEL = 70 mg/kg bw/day

Study Type / Animal / PMRA No.	Study Results
	59/70 mg/kg bw/day: ↓ pup wt/litter (F_1: PND14, F_2: PNDs 1 to 14) 177/208 mg/kg bw/day: ↓ pup wt/litter (F_1: PNDs 1-28, F_2: PNDs 1-21), pup bw (F_1: PNDs 1-28) and pup bwg (F_2: PNDs 1-21) 582/664 mg/kg bw/day: ↓ viability index (F_1), ↑ number of pups found dead between PNDs 2 and 4 (F_1 and F_2), ↓ pup wt/litter (F_1: PNDs 1-28, F_2: PNDs 1-21) and pup bwg (F_1: PNDs 1-28, F_2: PNDs 1-21)
Two-Generation Dietary Reproduction Toxicity Study Sprague-Dawley rats PMRA Nos. 1143142, 1210990 and 1874093	**Parental Toxicity** NOAEL = 80/76 mg/kg bw/day 255/228 mg/kg bw/day: ↓ bwg (F_0); ↓ bwg (F_1: ♂); ↓ bw (F_0 and F_1), ↑ liver (F_0 and F_1) and spleen (F_1) wts, yellow pigment in uterine suspensory ligament (F_0) (♀) **Reproductive Toxicity** NOAEL = 80/76 mg/kg bw/day 255/228 mg/kg bw/day: ↓ number of offspring born and alive one day following birth (F_{1b}), slightly ↓ litter sizes and litter wts (F_{1b}, F_{2a} and F_{2b}) **Offspring Toxicity** NOAEL = 80/76 mg/kg bw/day 255/228 mg/kg bw/day: ↓ number of offspring born and alive one day following birth (F_{1b}), slightly ↓ litter sizes and litter wts (F_{1b}, F_{2a} and F_{2b}), slightly higher incidence of small pups (F_{2b}), sinusoidal chronic inflammatory cells in the liver (F_2), ↓ bwg (F_{2b}), ↑ relative liver wt (F_2); ↓ bw (F_2) (♂)
Developmental Toxicity Study Sprague-Dawley rats PMRA Nos. 1142585, 1143143, 1214826 and 1874095	**Maternal Toxicity** NOAEL = 1000 mg/kg bw/day 3000 mg/kg bw/day: ↓ bw (GD15), bwg and fc, ↑ water intake **Developmental Toxicity** NOAEL = 1000 mg/kg bw/day 3000 mg/kg bw/day: ↓ fetal bw, increased incidence of small fetuses, slight dilation of the brain ventricles and the space between the body wall and organs (sign of immaturity), ↑ percentage of incomplete ossification of the caudal vertebrae
Range-finding Developmental Toxicity Study New Zealand White rabbits PMRA No. 1227063	No NOAEL established (range-finding) Maternal toxicity noted at ≥ 500 mg/kg bw/day included clinical signs of toxicity (green staining in urogenital area and ↓ defecation), ↓ bwg and fc, ↑ number of mortalities and abortions Developmental toxicity noted at ≥ 500 mg/kg bw/day included ↓ fetal bw and ↑ number of abortions
Developmental Toxicity Study New Zealand White rabbits PMRA Nos. 1227068, 1874095 and 1874093	**Maternal Toxicity** NOAEL =100 mg/kg bw/day 300 mg/kg bw/day: 1 mortality (GD20), ↓ bwg (GD 7-19), weight loss (GD 7-10) and ↓ fc 500 mg/kg bw/day: clinical signs of toxicity (green stained urogenital fur, ↓ defecation and urination), ↓ fc (GD 19-23), ↑ weight loss (GD 7-19) and ↑ number of abortions

Study Type / Animal / PMRA No.	Study Results
	Developmental Toxicity NOAEL = 300 mg/kg bw/day 500 mg/kg bw/day: umbilical herniation of the intestines and a rudimentary left atrium (1 fetus), hydrocephaly (4 fetuses (5.2% of fetuses) from 3 separate litters (27.3% of litters)
Reverse mutation assay *Salmonella typhimurium* (TA1538 and TA1978), *Escherichia coli* (W3623 pol- and wildtype) and *Bacillus subtilis* (H17 and M45) PMRA No. 2126780	Negative
Reverse mutation assay *Salmonella typhimurium* (TA98, TA100, TA1535, TA1537 and TA1538) and *Escherichia coli* (WP-2 uvrA) PMRA No. 2126773 and 1874093	Negative
Chromosomal aberrations Chinese hamster ovary cells (CHO-K1) PMRA No. 1143144 and 1874093	Negative
Chromosomal aberrations Chinese hamster ovary cells (CHO-K1) PMRA No. 1143145 and 1874093	Negative
Unscheduled DNA Synthesis Human Cells (HeLaS3) PMRA No. 2126775 and 1874093	Negative
Chromosomal Aberrations Bone marrow cells of ICR mice PMRA No. 2126777 and 1874093	Negative

Study Type / Animal / PMRA No.	Study Results
Acute Range-finding Neurotoxicity Study Wistar rats PMRA No. 2050133	No NOAEL established (range-finding). No evidence of neurotoxicity.
Acute Neurotoxicity Study Wistar rats PMRA Nos. 2126795 and 2050131	NOAEL = 2000 mg/kg bw/day (HDT). No evidence of neurotoxicity.
13-Week Dietary Neurotoxicity Study Wistar rats PMRA Nos. 2126797 and 2050134	NOAEL = 727/230 mg/kg bw/day (♂/♀). 739 mg/kg bw/day: ↓ bw and bwg (♂). 1456/1502 mg/kg bw/day: ↓ fc; ↓ bwg (♂); ↓ bw (♂). No evidence of neurotoxicity.
In Vitro Metabolism Study Various strains of animals PMRA No. 2221847	**Without NADPH** The guinea pig liver preparation was most active in degrading d-trans-phenothrin, followed by the dog, rabbit, rat and mouse. In all species tested, the major metabolite identified was 3-phenoxybenzyl alcohol. Smaller amounts of 3-phenoxybenzoic acid and a trace amount of 3-(4'-hydroxy) phenoxybenzoic acid were also formed. **With NADPH** The percent degradation of d-trans-phenothrin was not affected by the addition of NADPH except in the dog. Addition of NADPH gave rise to a decrease in 3-phenoxybenzyl alcohol with an accompanying increase in 3-phenoxybenzoic acid and unidentified ether-soluble metabolites. The formation of 3-(4'-hydroxy)phenoxybenzoic acid was not affected by NADPH.
In Vivo Uterotrophic and Hershberger Assays Sprague-Dawley rats PMRA No. 2221852	**Uterotrophic Assay** No treatment-related effects noted on clinical signs, body weight, food consumption, kidney or uterine weights. 1000 mg/kg bw/day: ↑ liver wts **Hershberger Assay** No treatment-related effects noted, in the presence or absence of testosterone propionate, on clinical signs of toxicity, body weight, food consumption, serum androgen levels, kidney weights or on the weights of the accessory glands and/or tissues (such as ventral prostate, dorso-lateral prostate, seminal vesicles with coagulating glands, levator ani plus bulbocavernosus muscles, glans penis and Cowper's glands). ≥ 300 mg/kg bw/day: ↑ liver wts Negative.

Table 2 Toxicology Endpoints for Use in Health Risk Assessment for d-Phenothrin

Exposure Scenario	Study	Point of Departure and Endpoint	CAF [1] or Target MOE
Acute dietary	Oral (gavage) developmental toxicity study – rabbits	NOAEL = 100 mg/kg bw/day (weight loss, ↓ body weight gain and food consumption)	100
	ARfD = 1.0 mg/kg bw		
Repeated dietary	12-month dietary toxicity study – dogs	NOAEL = 7.1 mg/kg bw/day (effects on pituitary (↑ and ↑), adrenal glands, liver and epididymides (↑))	100
	ADI = 0.07 mg/kg bw/day		
Short- and Intermediate-term dermal	21-day dermal toxicity study – rats	NOAEL = 1000 mg/kg bw/day (HDT)	100
Long-term dermal [2]	12-month dietary toxicity study – dogs	NOAEL = 7.1 mg/kg bw/day (effects on pituitary (↑ and ↑), adrenal glands, liver and epididymides (↑))	100
Short- and Intermediate-term inhalation	90-day inhalation toxicity study – rats	NOAEL = 26.6 mg/kg bw/day (eosinophilic inclusions in the olfactory epithelial cells of the nasal turbinates)	100
Long-term inhalation [3]	12-month dietary toxicity study – dogs	NOAEL = 7.1 mg/kg bw/day (effects on pituitary (↑ and ↑), adrenal glands, liver and epididymides (↑))	100
Non-dietary oral ingestion	Oral (gavage) developmental toxicity study – rabbits	NOAEL = 100 mg/kg bw/day (weight loss, ↓ body weight gain and food consumption)	100
Long-term Aggregate – Oral, Dermal and Inhalation	12-month dietary toxicity study – dogs	NOAEL = 7.1 mg/kg bw/day (effects on pituitary (↑ and ↑), adrenal glands, liver and epididymides (↑))	100
Cancer	Not required		

[1] CAF refers to a total of uncertainty and PCPA factors for dietary assessments. MOE refers to a target MOE for occupational and residential assessments.

[2] Since an oral NOAEL was selected, a dermal absorption factor of 17% was used in a route-to-route extrapolation.

[3] Since an oral NOAEL was selected, an inhalation absorption factor of 100% (default value) was used in route-to-route extrapolation.

Appendix IV Occupational and Residential Applicator and Postapplication Risk Assessment

Table 1 Short- to Intermediate-term Commercial Applicator Exposure and Risk Assessment

Site	Formulation [1]	Application Equipment	PPE [2]	Application Rate [3] (kg ai/acre)	LADD (d ai/day) [4]	Dermal Exposure [5] (mg/kg bw/day)	Dermal MOE [6]	Inhalation Exposure [7] (mg/kg bw/day)	Inhalation MOE [8]
Spot, crack and crevice indoor use only in homes and non-food areas of restaurants, schools, nursing homes, warehouses, offices, hotels, motels, kennels and hospitals Hornet, wasp and yellow jacket nests	EP (0.4% a.i.)	Aerosol	Baseline, no gloves	0.00218	1.0	0.10	4,500	6.51E-04	769,000

[1] EP = Pressurized product

[2] PPE = Personal protective equipment. Baseline (BS) = long-sleeved shirt, long pants and no chemical-resistant gloves

[3] No application rate (AR) was not provided in the SUL. The percent guarantee was used along with the can size to determine a rate in kg ai/ac (an [target registered] container size of 600 g with a 0.4% guarantee = 2.4 g ai/ac can).

[4] LADD = Lifetime average daily dose. Assumed based on professional judgment and other OPMSB risk assessments (approved Interscide) assuming two containers/home and a commercial applicator being able to treat seven homes/day (USEPA, 2006; Scientific, 2010).

[5] Where dermal exposure (mg/kg/day) = unit exposure × (AR) mg/kg × area treated/per day × application rate/100 kg.

[6] MOE = Margin of exposure. Dermal MOE = dermal NOAEL/dermal exposure. Based on a dermal NOAEL of 1000 mg/kg/bw/day and a target MOE of 100 applicable to short- and intermediate-term scenarios.

[7] Where inhalation exposure (mg/kg/bw/day) = unit exposure × (AR) mg/kg × area treated/per day × application rate/80 kg.

[8] MOE = margin of exposure. Inhalation MOE = inhalation NOAEL/inhalation exposure. Based on an inhalation NOAEL of 50.0 mg/kg/bw/day and a target MOE of 100 applicable to short- and intermediate-term scenarios.

Table 2 Short-term Residential Applicator Exposure Risk Assessment

Scenario	Formulation [1]	Application Equipment/ Method	Type	Application Rate (kg a.i./unit or gal) [2]	Amount Handled Daily [3]	Dermal Exposure (mg/kg bw/day) [4]	Dermal MOE [5]	Inhalation Exposure (mg/kg bw/day) [6]	Inhalation MOE [7]
Indoor environment	DU	Shaker can	Broadcast	0.0025	1	0.30	3400	1.2E-03	21 000
			Perimeter/spot	0.0025	0.5	0.15	6800	6.2E-04	43 000
	PP	Aerosol can	Broadcast surface spray	0.00118	1	0.032	31 000	2.6E-04	800 000
			Perimeter/spot/bed bug, crack and crevice (coarse and pin stream application)	0.00118	0.5	0.016	62 000	1.3E-04	200 000
			Space spray	0.00118	0.25	0.0081	120 000	6.6E-05	400 000
Outdoors and trees, and stinging insect nests			Contact spray	0.00118	2	0.065	15 000	5.3E-04	51 000
Treated pets	9%	Spot-on	Spot-on application	0.005211	2	0.035	29 000	Negligible	

[1] DU = dust or powder, PP = pressurized product, SN = solution

[2] Based on percent guarantee, and size and/or density of product or application rate provided on the label, if available. The maximum application rate was used for each scenario.

[3] Based on US EPA Residential SOPs (2012) measured in containers for DU and PP formulations and number of pets treated for SN formulations.

[4] Where dermal exposure (mg/kg bw/day) = (Unit Exposure) × Application Rate × Amount Handled per Day/80 kg. Dermal absorption is not required because the dermal NOAEL is based on a dermal toxicity study.

[5] MOE = Margin of exposure, MOE = NOAEL/Exposure, based on a dermal NOAEL of 1000 mg/kg bw/day and a target MOE of 100 applicable to short-term scenarios.

[6] Where inhalation exposure (mg/kg bw/day) = (unit exposure) × Application Rate × Amount Handled per day/80 kg.

[7] MOE = Margin of exposure, MOE = NOAEL/Exposure, based on an inhalation NOAEL of 26.6 mg/kg bw/day and a target MOE of 100 applicable to short-term scenarios.

Table 3 Short-term Postapplication Dermal Exposure from Floor and Carpets

Exposure Scenario		Life Stage	Transferrable Residue ($\mu g/cm^2$) [1]	Transfer Coefficient (cm^2/hr) [2]	Exposure Time (hr/day) [3]	Dermal Dose (mg/kg bw/day) [4]	MOE [5]
Broadcast	Carpet	Adults	1.362	6800	8	0.93	1100
		Youth		9600	5	0.67	1500
		Children		3800	4	0.89	1100
	Hard surface	Adults	1.816	6800	2	0.31	3200
		Youth		9600	1	0.18	5600
		Children		3800	2	0.59	1700
Perimeter/ spot/bed bug	Carpet	Adults	0.681	6800	8	0.46	2200
		Youth		9600	5	0.33	3000

Exposure Scenario		Life Stage	Transferable Residue (µg/cm²) [1]	Transfer Coefficient (cm²/hr) [2]	Exposure Time (hr/day) [3]	Dermal Dose (mg/kg bw/day) [4]	MOE [5]
(coarse and pinstream)		Children		1800	4	0.45	2200
	Hard surface	Adults	0.908	6800	2	0.15	6500
		Youth		5600	1	0.089	11 000
		Children		1800	2	0.30	3400
Crack and crevice	Carpet	Adults	0.1362	6800	8	0.093	11 000
		Youth		5600	5	0.067	15 000
		Children		1800	4	0.089	11 000
	Hard surface	Adults	0.1816	6800	2	0.031	32 000
		Youth		5600	1	0.018	56 000
		Children		1800	2	0.059	17 000
Space spray	Carpet	Adults	0.0141	6800	8	0.0096	100 000
		Youth		5600	5	0.0069	140 000
		Children		1800	4	0.0092	110 000
	Hard surface	Adults	0.0188	6800	2	0.0032	310 000
		Youth		5600	1	0.0018	540 000
		Children		1800	2	0.0062	160 000

[1] Where Transferable Residue (µg/cm²) = Residue (µg/cm²) × Fraction Transferred (%). Deposited residues were calculated based on the maximum label application rates or the calculated amount applied using the USEPA Residential SOPs (2012) algorithms for all scenarios.

[2] Transfer Coefficient (cm²/hr) default values obtained from the USEPA Residential SOPs (2012).

[3] Exposure Time (hr/day) default values obtained from the USEPA Residential SOPs (2012).

[4] Where Dermal Dose (mg/kg bw/day) = (Transferable Residue (µg/cm²) × 0.001 mg/µg × Transfer Coefficient (cm²/hr) × Exposure Time (hr/day))/Body Weight (kg). Body weights of 80, 57 and 11 kg were used for adults, youths (11 < 16 years), and children (1 < 2 years) respectively, as stated in the USEPA Residential SOPs (2012). Dermal absorption is not required because the dermal NOAEL is based on a dermal toxicity study.

[5] MOE = margin of exposure; MOE = NOAEL/exposure, based on a dermal NOAEL of 1000 mg/kg bw/day, and a target MOE of 100 applicable to short-term scenarios.

Table 4 Short-term Postapplication Dermal Exposure from Mattresses

Exposure Scenario	Life Stage	Deposited Residue ($\mu g/cm^2$) [1]	Surface Area/Body Weight Ratio (cm^2/kg) [2]	Dermal Dose (mg/kg bw/day) [3]	MOE [4]
Application to mattress	Adults	4.5	280	0.019	53 000
	Youth	4.5	280	0.019	53 000
	Children	4.5	640	0.043	23 000

[1] Default deposited residue value was obtained from the USEPA Residential SOPs (2012) since an application rate was not provided for products applied to mattresses.
[2] Values were obtained from the USEPA Residential SOPs (2012) based on body weights of 80 kg for adults, 57 kg for youth, and 11 kg for children (1 < 2 years).
[3] Where Dermal Dose (mg/kg bw/day) = (Deposited Residue ($\mu g/cm^2$) × 0.001 mg/µg × Surface Area/Body Weight Ratio (cm^2/kg) × Fraction of skin in contact with mattress (0.5) × Fraction transferred (0.06) × Protection Factor (0.5). Dermal absorption is not required because the dermal NOAEL is based on a dermal toxicity study.
[4] MOE = margin of exposure; MOE = NOAEL/exposure, based on a dermal NOAEL of 1000 mg/kg bw/day and a target MOE of 100 applicable to short-term scenarios.

Table 5 Short-term Postapplication Dermal Exposure from Gardens, Trees and Indoor Plants[1]

Exposure Scenario	Life Stage	DFR_t (ug/cm^2) [2]	Transfer Coefficient (cm^2/hr) [3]	Exposure Time (hour) [4]	Dermal Dose (mg/kg bw/day) [5]	MOE [6]
Gardens	Adults	0.661	8400	2.2	0.153	6500
	Youth		6900	2.2	0.176	5700
	Children		4600	1.1	0.105	9600
Trees and Retail Plants	Adults		1700	1	0.014	71 000
	Youth		1400	1	0.016	62 000
	Children		930	0.5	0.010	100 000
Indoor Plants	Adults	0.538	220	1	0.001	680 000
	Youth	0.538	180	1	0.002	590 000
	Children	0.538	120	0.5	0.001	990 000

[1] The risk assessment was conducted without chemical-specific DFR since no studies were provided. Default values obtained from USEPA Residential SOPs (2012).
[2] Where DFR_t = Application Rate (kg ai/ha) × Transferrable a.i. (0.25) × (1-(Dissipated Residue (0.1))^t (day after application (0)) × 1.0E09 ug/kg × 1.0 E-08 ha/cm^2. Based on two applications two weeks apart.
[3] Transfer Coefficient (cm^2/hr) default values obtained from the USEPA Residential SOPs (2012).
[4] Exposure Time (hr/day) default values obtained from the USEPA Residential SOPs (2012).
[5] Where Dermal Dose (mg/kg bw/day) = (DFR_t ($\mu g/cm^2$) × 0.001 mg/µg × Transfer Coefficient (cm^2/hr) × Exposure Time (hr)/Body Weight (kg). Body weights of 80, 57 and 32 kg were used for adults, youths and children (6 < 11 years) as stated in USEPA Residential SOPs (2012). Dermal absorption is not required because the dermal NOAEL is based on a dermal toxicity study.
[6] MOE = Margin of exposure; MOE = NOAEL/Exposure, based on a dermal NOAEL of 1000 mg/kg bw/day and a target MOE of 100 applicable to short-term scenarios.

Table 6 Short-term Postapplication Dermal Exposure from Treated Pets

Exposure Scenario		Life Stage	Surface Area of Pet (cm^2/animal) [1]	Transferable Residue (mg/cm^2) [2]	Transfer Coefficient [3] (cm^2/hr)	Exposure Time [4] (hours/day)	Dermal Dose [5] (mg/kg bw/day)	MOE [6]
Dog	Small (2.5-5 kg)	Adults	1989	5.79E-03	5200	0.77	0.29	3400
		Youth			4300	0.92	0.40	2500
		Children			1400	1	0.74	1400
	Medium (6-14 kg)	Adults	3513	6.56E-03	5200	0.77	0.33	3000
		Youth			4300	0.92	0.46	2200
		Children			1400	1	0.84	1200
	Large (14-28 kg)	Adults	6094	0.012	5200	0.77	0.60	1700
		Youth			4300	0.92	0.83	1200
		Children			1400	1	1.52	660
	Extra Large (> 28 kg)	Adults	9562	0.011	5200	0.77	0.55	1800
		Youth			4300	0.92	0.76	1300
		Children			1400	1	1.39	720

[1] Where Surface Area (cm^2) = ((12.3*((BW (kg)*1000 g/kg)^0.65)) as stated in the USEPA Residential SOPs (2012).
[2] Where Transferable Residue (mg/cm^2) = Application Rate (mg a.i./pet) × Fraction of application rate transferred (0.02)/ Surface area of pet cm^2/pet.
[3] Transfer Coefficient (cm^2/hr) default values obtained from the USEPA Residential SOPs (2012).
[4] Exposure Time (hr/day) default values obtained from the USEPA Residential SOPs (2012).
[5] Where Dermal Dose (mg/kg bw/day) = (Transferable Residue (mg/cm^2) × Transfer Coefficient (cm^2/hr) × Exposure Time (hour/day))/Body Weight (kg). Body weights of 80, 57 and 11 kg were used for adults, youths, and children (1 < 2 yrs) as stated in USEPA Residential SOPs (2012). Dermal absorption is not required because the dermal NOAEL is based on a dermal toxicity study.
[6] MOE = margin of exposure; MOE = NOAEL/exposure, based on a dermal NOAEL of 1000 mg/kg bw/day and a target MOE of 100 applicable to short-term scenarios.

Table 7 Long-term Postapplication Dermal Exposure from Mattresses

Exposure Scenario	Life Stage	Deposited Residue ($\mu g/cm^2$) [1]	Surface Area/Body Weight Ratio (cm^2/kg) [2]	Dermal Dose (mg/kg bw/day) [3]	MOE [4]
Application to mattress	Adults	4.5	280	1.1E-03	6600
	Youth	4.5	280	1.1E-03	6600
	Children	4.5	640	2.4E-03	2900

[1] Default deposited residue value was obtained from the USEPA Residential SOPs (2012) since an application rate was not provided for products applied to mattresses.
[2] Values were obtained from the USEPA Residential SOPs (2012) based on body weights of 80 kg for adults, 57 kg for youth, and 11 kg for children (1 < 2 years).
[3] Where Dermal Dose (mg/kg bw/day) = Deposited Residue ($\mu g/cm^2$) × 0.001 mg/µg × Surface Area/Body Weight Ratio (cm^2/kg) × Fraction of skin in contact with mattress (0.5) × Fraction transferred (0.02) × Protection Factor (0.5) × DA (17%).
[4] MOE = margin of exposure; MOE = NOAEL/ exposure, based on an oral NOAEL of 7.1 mg/kg bw/day and a target MOE of 100 applicable to long-term dermal scenarios.

Table 8 Long-term Postapplication Dermal Exposure from Floor and Carpets

Exposure Scenario		Life Stage	Transferable Residue ($\mu g/cm^2$) [1]	Transfer Coefficient (cm^2/hr) [2]	Exposure Time (hr/day) [3]	Dermal Dose (mg/kg bw/day) [4]	MOE [5]
Broadcast/perimeter/ spot/bed bug (coarse)	Carpet	Adults	0.09	4700	8	7.2E-03	990
		Youth	0.09	3900	5	5.2E-03	1400
		Children	0.09	1300	4	7.2E-03	980
	Hard surface	Adults	0.135	4700	2	2.7E-03	2600
		Youth	0.135	3900	1	1.6E-03	4500
		Children	0.135	1300	2	5.4E-03	1300
Perimeter/ spot/bed bug (pinstream)	Carpet	Adults	0.022	4700	8	1.8E-03	4000
		Youth	0.022	3900	5	1.3E-03	5500
		Children	0.022	1300	4	1.8E-03	4000
	Hard surface	Adults	0.033	4700	2	6.6E-04	11 000
		Youth	0.033	3900	1	3.8E-04	18 000
		Children	0.033	1300	2	1.3E-03	5400
Crack and crevice	Carpet	Adults	0.006	4700	8	4.8E-04	15 000
		Youth	0.006	3900	5	3.5E-04	20 000
		Children	0.006	1300	4	4.8E-04	15 000
	Hard surface	Adults	0.009	4700	2	1.8E-04	39 000
		Youth	0.009	3900	1	1.0E-04	68 000
		Children	0.009	1300	2	3.6E-04	20 000

[1] Where Transferable Residue ($\mu g/cm^2$) = Residue ($\mu g/cm^2$) × Fraction Transferred (%). Deposited residues were calculated based on the default residues provided in the USEPA Residential SOPs (2012) for all scenarios. The fraction transferred is based on the 50th percentile values for long-term risk assessments.

[2] Transfer Coefficient (cm^2/hr) default values were obtained from the USEPA Residential SOPs (2012) and are based on the 50th percentile values for long-term risk assessments.

[3] Exposure Time (hr/day) default values obtained from the USEPA Residential SOPs (2012).

[4] Where Dermal Dose (mg/kg bw/day) = (Transferable Residue ($\mu g/cm^2$) × 0.001 mg/μg × Transfer Coefficient (cm^2/hr) × Exposure Time (hr/day) × DA (17%))/Body Weight (kg). Body weights of 80, 57 and 11 kg were used for adults, youths, and children (1 < 2 years) respectively, as stated in the USEPA Residential SOPs (2012).

[5] MOE = margin of exposure; MOE = NOAEL/exposure, based on an oral NOAEL of 7.1 mg/kg bw/day and a target MOE of 100 applicable to long-term dermal scenarios.

Table 9 Short-term Postapplication Inhalation Exposure from Indoor Space Sprays

Exposure Scenario	Life Stage	Initial Concentration, C_0 (mg/m^3) [1]	Inhalation Dose (mg/kg bw/day) [2]	MOE [3]
Space spray	Adults	1.29	0.014	2000
	Youth		0.019	1400
	Children		0.051	520

[1] Initial Concentration (mg/m^3) = Application Rate (kg a.i./m^3) × 1.00E06 mg/kg. Application Rate was calculated using: $R = \frac{A.I. \times AA \times CF1 \times CF2}{V_{room}}$

Where *a.i. is amount of active ingredient (g/can) using largest container size (600 g) and highest guarantee (0.20%), AA = amount of product applied (fraction of can) calculated using 12 seconds of spray (label), 1.5 g/sec release rate, and 600 g container size, CF1 = 1.0E-06 kg/mg, CF2 = 1000 mg/g, V_{room} = 28 m^3 (box[sic] on label).*

[2] Inhalation Dose (mg/kg bw/day) = $\frac{C_0 \times IR \cdot [1-e^{(-ACH \times ET)}]}{ACH \times BW}$

Where IR = *Inhalation Rate (m^3/hour) 0.64, 0.63 and 0.33 m^3/hr for adult, youth and children (1 – 2 years old) respectively*; ACH = *Air Exchange per hour (0.45 hr^{-1})*; ET = *Exposure Time (2 hr)*; BW = *Body Weight (80 kg for adults, 57 kg for youth and 11 kg for children (1 – 2 years old). Default values were obtained from the USEPA Residential SOPs (2012).*

[3] MOE = *margin of exposure*; MOE = *NOAEL/Exposure, based on an inhalation NOAEL of 26.6 mg/kg bw/day and a total* MOE of 100 *applicable to short-term scenarios.*

Table 10 Short-term Postapplication Inhalation Exposure from Indoor Surface Directed Sprays

Exposure Scenario	Life Stage	Mass of a.i. (mg) [1]	Exposure Time (hour) [2]	Inhalation Dose (mg/kg bw/day) [3]	MOE [4]
Surface-directed spray	Adults	1180	16	2.7E-04	100 000
	Youth	1180	15	3.4E-04	78 000
	Children	1180	18	1.1E-03	23 000

[1] Where $M_{a.i.}$ = Application Rate (kg a.i./can) × Amount Handled (1 can) × 1.00E+06. Application rate calculated based on the largest container size and % a.i. guarantee (0.001918 kg a.i./can).

[2] Exposure Time (hr/day) default values obtained from the USEPA Residential SOPs (2012).

[3] Where inhalation exposure (mg/kg bw/day) = $\frac{IR \times M}{ACH \times V} \times \left\{ \left[\frac{(ACH \times e^{(k \times ET)}) - (k \times e^{-ACH \times ET})}{ACH - k} \right] - \frac{1}{ACH} \right\} \times \frac{1}{BW}$

The equation assumes 100% absorption through inhalation, air exchanges (ACH) = 0.45 hr^{-1}, volume of a room (V) = 28 m^3, decay rate (k) = 9.5E-06 hr^{-1}, M = mass of a.i., ET = exposure time. Inhalation rates (IR) of 0.64, 0.63 and 0.33 m^3/hr and body weights (BW) of 80, 57 and 11 kg were used for adults, youth and children (1 – 2 years old) respectively, as stated in the USEPA Residential SOPs (2012).

[4] MOE = *margin of exposure*; MOE = *NOAEL/Exposure, based on an inhalation NOAEL of 26.6 mg/kg bw/day and a total* MOE of 100 *applicable to short-term scenarios.*

Table 11 Long-term Postapplication Inhalation Exposure from Indoor Surface Directed Sprays

Exposure Scenario	Life Stage	Mass of a.i. (mg) [a]	Exposure Time (hours) [b]	Inhalation Dose (mg/kg bw/day) [c]	MOE [d]
Surface directed spray	Adults	[illegible]	[illegible]	[illegible]	[illegible]
	Youth	[illegible]	[illegible]	[illegible]	[illegible]
	Children	[illegible]	[illegible]	[illegible]	[illegible]

[a] Where $M_{a.i.}$ = (application frate (kg a.i./ unit) × (amount finished (1 unit) × 1,000 /lb). Application rate calculated based on the largest container size and % a.i. guarantee (0.003559 kg a.i./ unit).
[b] Exposure Time (hr/day) default values obtained from the US EPA's Residential SOPs (2012).
[c] Where inhalation exposure (mg/kg bw/day) = $\frac{MR \times M_{a.i.} \times}{AUR \times V}$ = [illegible] The equation assumes 100% absorption through inhalation, air exchanges (ACH) = 0.45 hr^{-1}, volume of a room (V) = 20 m^3, Room rate (R) = 0.58 m^3/hr^{-1}, M = mass of a.i., ET = exposure time, inhalation rates (IR) of 0.63, 0.63 and 0.53 m^3/hr and body weights (BW) of 80, 37 and 15 kg were used for adults, youth and children (3 – 5 years old) respectively, as stated in the US EPA's Residential SOPs (2012).
[d] MOE = margin of exposure, MOE = NOAEL/Exposure, based on an oral NOAEL of 7.1 mg/kg bw/day and a target MOE of 100 applied to long-term inhalation scenarios.

Table 12 Short-term Postapplication Hand-to-Mouth Exposure to Children from Indoor Environments

Exposure Scenario		Hand Residue Loading (mg/cm^2) [a]	Oral Dose (mg/kg bw/day) [b]	MOE [c]
Broadcast	Carpet	[illegible]	[illegible]	[illegible]
	Hard Surface	[illegible]	[illegible]	[illegible]
Perimeter/spot/bed bug (crevice and pin stream)	Carpet	[illegible]	[illegible]	[illegible]
	Hard Surface	[illegible]	[illegible]	[illegible]
Couch and crevice	Carpet	[illegible]	[illegible]	[illegible]
	Hard Surface	[illegible]	[illegible]	[illegible]
Space spray	Carpet	[illegible]	[illegible]	[illegible]
	Hard Surface	[illegible]	[illegible]	[illegible]

[a] Based on the dermal postapplication exposure from indoor applications without the body weight – fraction of a.i. on hands compared to body (0.15).
[b] Where Oral Dose (mg/kg bw/day) = [Hand Residue (mg/cm^2)] × [Fraction of hand mouthed/event (0.15) × Surface Area of one hand (320 cm^2)] × [Exposure Time (hr) × Replenishment Intervals (times) × [1 – (1 – Saliva Extraction Factor (0.46))$^{\text{Number of events per hour} \times \text{Exposure time (hr)}}$]] ÷ Body Weight (15 kg). Exposure times for carpets and hard surfaces were 4 and 2 hours, respectively, as stated in the US EPA's Residential SOPs (2012).
[c] MOE = margin of exposure, Oral MOE = oral NOAEL/Oral exposure, based on an oral NOAEL of 100 mg/kg bw/day and a target MOE of 100.

Table 13 Short-term Postapplication Object-to-Mouth Exposure to Children from Indoor Environments

Exposure Scenario		Object Residue (ug/cm^2) [1]	Oral Dose (mg/kg bw/day) [2]	MOE [3]
Broadcast	Carpet	1.367	0.018	5600
	Hard surface	1.816	0.017	5800
Perimeter/spot/bed bug (coarse and pin stream)	Carpet	0.681	0.009	11 000
	Hard surface	0.908	0.008	17 000
Crack and crevice	Carpet	0.136	0.002	56 000
	Hard surface	0.182	0.001	84 000
Space spray	Carpet	0.014	0.0002	540 000
	Hard surface	0.019	0.0001	810 000

[1] Where: Object Residue = Deposited Residue (ug/cm^2) × Fraction of residue transferred (6% for carpets and 8% for hard surfaces). Deposited residue based on maximum application rate provided on the labels.

[2] Where: Oral Dose (mg/kg bw/day) = [Object Residue (ug/cm^2) × 0.001 mg/ug × Surface Area of object mouthed (10 cm^2/event) × (Exposure Time (hr) × Replenishment Intervals (4/hr)) × (1 – (1 – Saliva Extraction Factor (0.48))^[Number of min per hour / (Oral/gastrointestinal transfer (20hr))]] @ Body Weight (11 kg). Exposure times for carpets and hard surfaces were 4 and 2 hours, respectively as stated in the USEPA Residential SOPs (2012).

[3] MOE = margin of exposure, Oral MOE = oral NOAEL/Oral exposure, based on an oral NOAEL of 100 mg/kg bw/day and a target MOE of 100.

Table 14 Short-term Postapplication Hand-to-Mouth Exposure to Children from Treated Pets

Exposure Scenario	Animal Size (kg)	Hand Residue Loading (mg/cm^2) [1]	Oral Dose (mg/kg bw/day) [2]	MOE [3]
Dog	2.5–5	0.0011	0.0074	14 000
	6–14	0.0012	0.0084	12 000
	14–28	0.0022	0.015	6600
	> 28	0.0020	0.014	7200

[1] Based the postapplication dermal exposure from spot-on applications without the body weight × fraction of a.i. on hands compared to body (0.04).

[2] Where Oral Dose (mg/kg bw/day) = (Hand Residue (mg/cm^2) × (Fraction of hand mouthed/event (0.11) × Surface Area of one hand (150 cm^2)) × (Exposure Time (hr) × Replenishment Intervals (4/hr)) × (1 – (1 – Saliva Extraction Factor (0.48))^[Number of min per hour / (Oral/gastrointestinal transfer (20hr))]] @ Body Weight (11 kg). Exposure time of 1 hour as stated in the USEPA Residential SOPs (2012).

[3] MOE = Margin of Exposure, Oral MOE = Oral NOAEL/Oral Exposure, based on an Oral NOAEL of 100 mg/kg bw/day and a target MOE of 100.

Table 15 Long-term Postapplication Hand-to-Mouth Exposure to Children from Indoor Environments

Exposure Scenario		Hand Residue Loading (mg/cm^2) [1]	Oral Dose (mg/kg bw/day) [2]	MOE [3]
Broadcast/perimeter/spot/bed bug (course)	Carpet	2.34E-04	5.4E-03	1300
	Hard surface	1.76E-04	2.0E-03	3500
Perimeter/spot/bed bug (pin stream)	Carpet	5.72E-05	1.3E-03	5400
	Hard surface	4.29E-05	5.0E-04	14 000
Crack and crevice	Carpet	1.56E-05	3.6E-04	20 000
	Hard surface	1.17E-05	1.4E-04	52 000

[1] Based the dermal postapplication exposure from indoor applications without the body weight × fraction of a.i. on hands compared to body (0.15).
[2] Where Oral Dose (mg/kg bw/day) = (Hand Residue (mg/cm^2) × (Fraction of hand mouthed/event (0.12) × Surface Area of one hand (150 cm^2)) × (Exposure Time (hr) × Replenishment Intervals (4/hr)) × (1 – (1 – Saliva Extraction Factor (0.48)) $^{\text{Number events per hour (1/Replenishment Intervals (4hr))}}$)]/ Body Weight (11 kg). Exposure times for carpets and hard surfaces were 4, and 2 hours, respectively, as stated in the USEPA Residential SOPs (2012).
[3] MOE = margin of exposure; Oral MOE = oral NOAEL/Oral exposure, based on an oral NOAEL 7.1 mg/kg bw/day and a target MOE of 100.

Table 16 Long-term Postapplication Object-to-Mouth Exposure to Children from Indoor Environments

Exposure Scenario		Object Residue (ug/cm^2) [1]	Oral Dose (mg/kg bw/day) [2]	MOE [3]
Broadcast	Carpet	0.090	1.1E-03	6300
	Hard surface	0.135	8.4E-04	8400
Perimeter/spot/bed bug (course and pin stream)	Carpet	0.022	2.8E-04	26 000
	Hard surface	0.033	2.1E-04	34 000
Crack and crevice	Carpet	0.006	7.5E-05	95 000
	Hard surface	0.009	5.6E-05	130 000

[1] Where Object Residue = Deposited Residue (ug/cm^2) × Fraction of residue transferred (2% for carpets and 3% for hard surfaces). Deposited residue based on default residues provided in the USEPA Residential SOPs (2012).
[2] Where Oral Dose (mg/kg bw/day) = [Object Residue (ug/cm^2) × 0.001 mg/ug x Surface Area of object mouthed (10 cm^2/event) × (Exposure Time (hr) x Replenishment Intervals (4/hr)) × (1 – (1 – Saliva Extraction Factor (0.48)) $^{\text{Number events per hour (1/Replenishment Intervals (4hr))}}$)]/ Body Weight (11 kg). Exposure times for carpets and hard surfaces were 4 and 2 hours, respectively as stated in the USEPA Residential SOPs (2012).
[3] MOE = margin of exposure; Oral MOE = oral NOAEL/Oral exposure, based on an oral NOAEL 7.1 mg/kg bw/day and a target MOE of 100.

Table 17 Short-term Dermal and Inhalation Aggregate for Applicator and Postapplication Exposure for Adults

Scenario	Formulation	Application Equipment/ Method	Type	Applicator Dermal MOE [1]	Postapplication Dermal MOE [2]	Aggregate Dermal MOE [3]	Applicator Inhalation MOE [1]	Postapplication Inhalation MOE [4]	Aggregate Inhalation MOE [3]
Indoor environment	Dust/powder	Shaker can	Broadcast	3400	1100	820	21 000	100 000	18 000
			Broadcast/ perimeter/spot	6800	1100	930	43 000	100 000	30 000
	Pressurized product	Aerosol can	Broadcast surface spray	31 000	1100	1000	100 000	100 000	50 000
			Perimeter/spot/ bed bug (coarse, pin stream); crack and crevice	62 000	2200	2100	200 000	100 000	67 000
			Mattress spray	62 000	53 000	29 000	200 000	100 000	67 000
			Space spray	120 000	100 000	56 000	400 000	2000	2000
Gardens/ trees			Contact spray	15 000	6500	4600	51 000	Negligible	NA
Treated pets	Solution	Spot-on	Spot-on	29 000	1700	1600	Negligible	Negligible	NA

[1] Based on MOE values from Table 2.

[2] Based on MOE values from Tables 3 (Indoor Environments), 4 (Mattresses), 5 (Gardens and Trees), and 6 (Treated Pets). The lowest postapplication MOEs for each scenario were used as a conservative estimate of aggregate exposure.

[3] Where Combined MOE = $\frac{1}{[(1/MOE_{app}) + (1/MOE_{postapp})]}$

[4] Based on MOE values from Tables 9 (Space Sprays) and 10 (Surface Sprays). Garden and trees inhalation exposure is considered to be minimal due to the low vapour pressure and outdoor application. Treated pet inhalation exposure is considered minimal based on the USEPA Residential SOPs (2012).

NA = not applicable.

Table 18 Short-term Postapplication Incidental Oral and Dietary Aggregate Exposure for Children (1 < 2 years)

Exposure Scenario	Oral Dose (mg/kg bw/day) [1, 2]	Dietary Dose (mg/kg bw/day) [3]	Aggregate Oral Dose (mg/kg bw/day) [4]	Aggregate Oral MOE [5]
Indoor Scenarios				
Broadcast carpet	0.13	0.000552	0.1343	740
Broadcast hard surface	0.045	0.000552	0.0451	2200
Perimeter/spot/bed bug carpet	0.067	0.000552	0.0674	1500
Perimeter/spot/bed bug hard surface	0.022	0.000552	0.0228	4400
Crack and crevice carpet	0.013	0.000552	0.0139	7200
Crack and crevice hard surface	0.0045	0.000552	0.0050	20 000
Space spray carpet	0.0014	0.000552	0.0019	52 000
Space spray hard surface	0.0005	0.000552	0.0010	99 000
Treated Pets				
Treated pets (small dog)	0.0074	0.000552	0.0079	13 000
Treated pets (medium dog)	0.0084	0.000552	0.0089	11 000
Treated pets (large dog)	0.015	0.000552	0.0158	6300
Treated pets (extra-large dog)	0.014	0.000552	0.0145	6900

[1] Indoor scenario oral dose values from Table 12.
[2] Treated pet scenario oral dose values from Table 14.
[3] Based on background chronic dietary exposure for children (1 < 2 years old).
[4] Where Aggregate Oral Dose = Oral Dose + Dietary Dose.
[5] MOE = margin of exposure; Aggregate Oral MOE = Oral NOAEL/Aggregate oral exposure, based on an oral NOAEL 100 mg/kg bw/day and a target MOE of 100.

Table 19 Long-term Postapplication Inhalation, Dermal and Dietary Aggregate Exposure

Exposure Scenario	Lifestage	Inhalation Dose [1] (mg/kg bw/day)	Surface Dermal Dose [2] (mg/kg bw/day)	Mattress Dermal Dose [3] (mg/kg bw/day)	Incidental Oral Dose [4] (mg kg bw/day)	Dietary Dose [5] (mg/kg bw/day)	Aggregate Dose [6] (mg/kg bw/day)	Aggregate Oral MOE [7]
Indoor Scenarios								
Broadcast, perimeter/spot/bed bug (coarse) – soft surface	Adult	1.0E-04	7.2E-03	1.1E-03	—	1.8E-04	8.5E-03	830
	Youth	1.3E-04	5.2E-03	1.1E-03	—	1.8E-04	6.6E-03	1100
	Child (1 < 2 yrs)	4.3E-04	7.2E-03	2.4E-03	5.4E-03	5.5E-04	1.6E-02	440
Broadcast, perimeter/spot/bed bug (coarse) – hard surface	Adult	1.0E-04	2.7E-03	1.1E-03	—	1.8E-04	4.0E-03	1800
	Youth	1.3E-04	1.6E-03	1.1E-03	—	1.8E-04	2.9E-03	2400
	Child (1 < 2 yrs)	4.3E-04	5.4E-03	2.4E-03	2.0E-03	5.5E-04	1.1E-02	650
Perimeter/spot/bed	Adult	1.0E-04	1.8E-03	1.1E-03	—	1.8E-04	3.1E-03	2300

Exposure Scenario	Lifestage	Inhalation Dose [1] (mg/kg bw/day)	Surface Dermal Dose [2] (mg/kg bw/day)	Mattress Dermal Dose [3] (mg/kg bw/day)	Incidental Oral Dose [4] (mg kg bw/day)	Dietary Dose [5] (mg/kg bw/day)	Aggregate Dose [6] (mg/kg bw/day)	Aggregate Oral MOE [7]
bug (pin stream) – soft surface	Youth	1.3E-04	1.3E-03	1.1E-03	—	1.8E-04	2.7E-03	2700
	Child (1 < 2 yrs)	4.3E-04	1.8E-03	2.4E-03	1.3E-03	5.5E-04	6.5E-03	1100
Perimeter/spot/bed bug (pin stream) – hard surface	Adult	1.0E-04	6.6E-04	1.1E-03	—	1.8E-04	2.0E-03	3500
	Youth	1.3E-04	3.8E-04	1.1E-03	—	1.8E-04	1.8E-03	4000
	Child (1 < 2 yrs)	4.3E-04	1.3E-03	2.4E-03	5.0E-04	5.5E-04	5.3E-03	1400
Crack and crevice – soft surface	Adult	1.0E-04	4.8E-04	1.1E-03	—	1.8E-04	1.8E-03	3900
	Youth	1.3E-04	3.5E-04	1.1E-03	—	1.8E-04	1.7E-03	4100
	Child (1 < 2 yrs)	4.3E-04	4.8E-04	2.4E-03	3.6E-04	5.5E-04	4.3E-03	1700
Crack and crevice – hard surface	Adult	1.0E-04	1.8E-04	1.1E-03	—	1.8E-04	1.5E-03	4600
	Youth	1.3E-04	1.0E-04	1.1E-03	—	1.8E-04	1.5E-03	4800
	Child (1 < 2 yrs)	4.3E-04	3.6E-04	2.4E-03	1.4E-04	5.5E-04	3.9E-03	1800

[1] Long-term indoor surface directed spray inhalation dose values from Table 11.
[2] Long-term indoor surface dermal dose values from Table 8.
[3] Long-term mattress dermal dose values from Table 7.
[4] Long-term incidental oral dose from Table 15.
[5] Based on background chronic dietary exposure for the general population and children (1 < 2 years old).
[6] Where Aggregate Oral Dose = Inhalation Dose + Surface Dermal Dose + Mattress Dermal Dose + Dietary Dose + Incidental Oral (children 1 < 2 years old).
[7] MOE = margin of exposure; Aggregate MOE = Long-term NOAEL/Aggregate oral exposure, based on an oral NOAEL 7.1 mg/kg bw/day and a target MOE of 100 applicable to long-term aggregate scenarios.

Appendix V Dietary Exposure and Risk Estimates for d-Phenothrin

Table 1 Acute Dietary (Food + Water) Exposure and Risk Estimates

Acute Exposure and Risk	99.9th percentile	
Population	Exposure [1] (mg/kg/day)	%RfD (%)
General population	0.000255	0.5
All infants	0.000710	0.7
Children 1–2 yrs	0.000699	0.7
Children 3–5 yrs	0.000557	0.6
Children 6–12 yrs	0.000310	0.3
Youth 13–19 yrs	0.000183	0.2
Adults 20–49 yrs	0.000163	0.2
Adults 50+ yrs	0.000160	0.1
Females 13–49 yrs	0.000178	0.2

[1] Probabilistic evaluation at risk at the 99.9th percentile using the %RfD of 1.0 mg/kg/day (Monte Carlo iterations = 500; seed = 1).

Table 2 Chronic Dietary (Food + Water) Exposure and Risk Estimates

Population	Exposure [1] (mg/kg bw/day)	%RfD (%)
General population	0.000103	0.1
All infants	0.000255	0.3
Children 1–2 yrs	0.000255	0.3
Children 3–5 yrs	0.000187	0.2
Children 6–12 yrs	0.000120	0.1
Youth 13–19 yrs	0.000078	0.1
Adults 20–49 yrs	0.000071	0.1
Adults 50+ yrs	0.000070	0.1
Females 13–49 yrs	0.000076	0.1

[1] Evaluation of exposure using the %RfD of 0.07 mg/kg bw/day.

Appendix VI Residue Chemistry Summary

There are no uses of d-phenothrin for direct application to agricultural crops or other foods. The labels specify no usage in areas where food processing or preparation occurs. Therefore, exposure from food is not expected to occur and if so, it would be only through indirect application. Potential indirect dietary exposure may occur through consumption of imported foods from the United States, where d-phenothrin is used in mosquito abatement programs.

Maximum residue limits have not been specified for d-phenothrin in Canada. Where no specific maximum residue limit has been established, subsection B.15.002(1) of the Food and Drug Regulations applies, which requires that residues not exceed 0.1 ppm. In the United States, tolerances for d-phenothrin were set at 0.01 ppm on all commodities to cover indirect application on all food or feed crops, which may occur from mosquito abatement program. No CODEX maximum residue limits have been set for d-phenothrin.

Currently there is no residue definition in Canada for d-phenothrin. In the United States, the residue definition for enforcement and risk analysis is the sum of the 4 isomers of d-phenothrin: *[(3-phenoxyphenyl)methyl] 2,2-Dimethyl-3-(2-methyl-1-propenyl)cyclopropanecarboxylate]*, for both plants and animals.

Since d-phenothrin is not directly applied to foods in Canada, standard data requirements for food residue chemistry are not applicable. However, some information is available to understand the metabolism, analytical methodology and residues of d-phenothrin under current use conditions.

In rats, d-phenothrin is rapidly absorbed and excreted in the urine and feces. In plants, d-phenothrin is rapidly absorbed, converted into carboxylated forms, and then incorporated into cellular constituents.

Limited data is available on the analytical methodology. However, adequate multiresidue methods are available that are used by the United States Department of Agriculture's Pesticide Data Program (USDA-PDP) to measure d-phenothrin residues in foods and feeds. The Canadian Food Inspection Agency does not monitor for d-phenothrin.

The only residues expected from d-phenothrin use in Canada would be from applications in residences or commercial areas where food processing or food storage takes place. However, studies show that no residues occur when food commodities are covered during treatment. The assumption of zero food residues in domestic food is therefore acceptable to the PMRA provided that all labels explicitly prescribe removal or covering of food/feed commodities during application.

There is no significant risk from exposure to d-phenothrin from the Canadian diet.

Appendix VII Environmental Exposure and Risk Assessment for d-Phenothrin

Table 1 Fate and Behaviour in the Environment

Property	Test Substance	Value	Transformation Products	Comments	PMRA No.
Abiotic Transformation					
Hydrolysis	Technical	393 d @pH5 186 d @pH7 89 d @pH9	3-phenoxybenzyl alcohol	Persistent at pH5 -7 Moderately persistent at pH 9	1166311 1166359
Phototransformation on soil	—	—	—	No data available	—
Phototransformation in water	Technical	$t_{1/2}$ = 13.9 h	—	Non-persistent	1166362 1166363 2415668
Biotransformation					
Biotransformation in aerobic soil	Technical	$t_{1/2}$ = 26 d	—	Slightly persistent	116363 116367
Biotransformation in anaerobic soil			—	No data available	
Biotransformation in aerobic water		$t_{1/2}$ = 36.1 d	—	Slightly persistent	2415668
Biotransformation in anaerobic water		$t_{1/2}$ = 173.3 d	—	Moderately persistent	2415668
Mobility					
Adsorption / desorption in soil	—	K_{oc} = 141000	—	Immobile	2415668
Volatilization	—	1.9×10^{-2} mPa @ 21°C	—	Non-volatile	
Field studies					
Field dissipation	—	$t_{1/2}$ = 1-4 d	—	California & Georgia	1166372 1166379

Table 2 Toxicity to Non-Target Species

Organism	Exposure	Test Substance	Endpoint Value
Earthworm	14 d-acute	Technical	> 1000 mg/kg soil
Bee	Oral	—	No data available.
	96 h-contact	Technical	LD_{50} = 0.067 µg/bee
Predatory arthropod	Contact	—	No data available.
Parasitic arthropod	Contact	—	No data available.
Bobwhite quail	Acute	Technical	LD_{50} > 25 000 mg/kg
	5 d-dietary	Technical	LD_{50} > 5620 mg/kg
	Reproduction	—	No data available.
Rat	Acute	Technical	LD_{50} > 2000 mg/kg
	5 d-dietary	Technical	LD_{50} > 5620 mg/kg
	Reproduction	—	No data available.
Vascular plant	Seedling emergence	—	No data available.
	Vegetative vigour	—	No data available.
Daphnia magna	48 h-acute	Technical	0.0043 mg/L
	Chronic	—	NOEC = 0.47 µg/L

Organism	Exposure	Test Substance	Endpoint Value
Rainbow trout	96 h-acute	Technical	2.7 µg/L
	Chronic	—	No data available.
Bluegill sunfish	96 h-acute	Technical	16 µg/L
	Chronic	—	No data available.
Freshwater alga	Acute	—	No data available.
Vascular plant	Dissolved	—	No data available.

Table 3 Toxic Substances Management Policy Considerations – Comparison to TSMP Track 1 Criteria

TSMP Track 1 Criteria	TSMP Track 1 Criterion Value		Are Criteria Met for d-Phenothrin?
CEPA toxic or CEPA toxic equivalent[1]	Yes		Yes
Predominantly anthropogenic[2]	Yes		Yes
Persistence[3]	Soil	Half-life ≥ 182 days	No: Half-life = 26 d
	Water	Half-life ≥ 182 days	No: Half-life = 36.1 d
	Sediment	Half-life ≥ 365 days	Not available.
	Air	Half-life ≥ 2 days or evidence of long-range transport	Half-life or volatilization is not an important route of dissipation and long-range atmospheric transport is unlikely to occur based on the vapour pressure (1.9×10^{-2} mPa @ 21°C) and Henry's law constant (6.75×10^{-1} Pa·m^3/mol).
Bioaccumulation[4]	Log $K_{ow} \geq 5$		Yes: 6.01
	BCF ≥ 5000		No: < 4000
	BAF ≥ 5000		Not available
Is the chemical a TSMP Track 1 substance (all four criteria must be met)?			No, does not meet TSMP Track 1 criteria.

[1] All pesticides will be considered CEPA-toxic or CEPA-toxic equivalent for the purpose of initially assessing a pesticide against the TSMP criteria. Assessment of the CEPA toxicity criteria may be refined if required (all other TSMP criteria are met).
[2] The policy considers a substance "predominantly anthropogenic" if, based on expert judgment, its concentration in the environment medium is largely due to human activity, rather than to natural sources or releases.
[3] If the pesticide and/or the transformation product(s) meet one persistence criterion identified for one media (soil, water, sediment or air), then the criterion for persistence is considered to be met.
[4] The log K_{ow} and/or BCF and/or BAF are preferred over log K_{ow}.

Appendix VIII Label Amendments for End-Use Products Containing d-Phenothrin

The label amendments presented below do not include all label requirements for individual end-use products, such as first aid statements, disposal statements, precautionary statements and supplementary protective equipment. Information on labels of currently registered products should not be removed unless it contradicts the following label statements.

The following label statements are proposed to ensure consistency in label statements and with the assumptions used in the health risk assessment, minimize unnecessary exposure and further protect the environment.

I) The following statements are proposed to be included in a section entitled **DIRECTIONS FOR USE**

For all products:

- Keep foodstuff and food utensils out of room or covered during application and keep these off treated surfaces until treated area is vacuumed.

For all **dust** products registered for use indoors:

- Application as broadcast, perimeter/spot, and crack and crevice is permitted.

For all **indoor aerosol** products, except space sprays and wasp/hornet nest sprays:

- DO NOT apply as a broadcast application. ONLY perimeter/spot or crack and crevice is permitted. Perimeter/spot application is defined as an application in a wide band or strip around the perimeter of the room or over a small area (< 2 ft^2/0.2 m^2). Crack and crevice applications are defined as an application with the use of a pin stream nozzle, into cracks and crevices in which pests hide or through which they may enter a building. Application to upholstered furniture must be limited to tufts and seams or spot treatment.

For all products registered for use on **mattresses**:

- Before treatment, remove all bedding and thoroughly vacuum and air out mattresses and box springs. Treat mattresses, box springs, bed frames, and headboards, especially tufts, folds and edges of the mattress. Allow treatment to dry before remaking bed with freshly washed bedding.

For all products registered for use on **plants**:

- Not for use in greenhouses, vegetable gardens or on food-bearing plants.

II) The following statements are proposed to be included in a section entitled

ENVIRONMENTAL HAZARDS

For all products:

- Toxic to aquatic organisms.

For d-phenothrin products that are registered for outdoor use (not for products that are registered for indoor use only):

- Toxic to bees. Do not spray bees.

References

A. Studies Considered in the Chemistry Assessment

LIST OF STUDIES/INFORMATION SUBMITTED BY REGISTRANT

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B. Studies Considered in the Toxicological Assessment

LIST OF STUDIES/INFORMATION SUBMITTED BY REGISTRANT

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ADDITIONAL INFORMATION CONSIDERED

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